THE BRITISH JOURNAL

OF

OPHTHALMOLOGY

FEBRUARY, 1931

COMMUNICATIONS

HOFRAT PROFESSOR ERNST FUCHS AND HIS WORK

By the death of Hofrat Professor Ernst Fuchs from angina pectoris on November 19, 1930, in his 80th year, ophthalmology has lost its most lucid and able exponent, and ophthalmologists the world over, a most charming and loveable companion.

Fuchs was a pupil of the famous surgeon Billroth, and studied ophthalmology in Vienna under Professor Ferdinand Arlt, whose assistant he became and to whose chair at the University he subsequently succeeded.

The Vienna school of ophthalmology was the first to be formed in Europe on the renaissance of ophthalmology towards the close of the 18th century. Only last year, at the opening of the Wilmer Institute, Fuchs gave the following interesting account of its origin.

An itinerant coucher of cataracts, named Wenzel of Paris, "a completely ignorant fellow," obtained skill and reputation in his method of operating by practice on the highways of France, where he collected blind beggars and vagrants and relieved them of their cataracts. His fame spread so that the Austrian Empress Maria Theresa sent for him in 1775 to operate on one of the ladies of her court. Before leaving Vienna he was paid to impart the details of his technique to three young physicians there. One of them, a teacher of anatomy named Barth, was then commissioned to teach ophthalmology also. It was Barth's pupil Beer, who in 1812, became the first professor of ophthalmology in the University of Vienna and was given an independent clinic with two wards containing twelve beds each. Ferdinand Arlt, Fuchs's teacher, who succeeded Beer in the post was a self-made man of humble

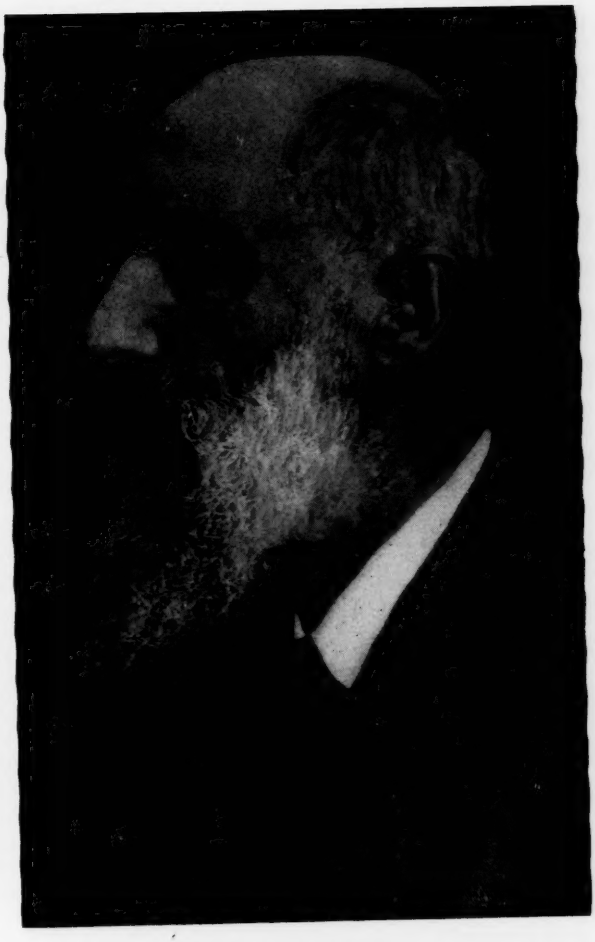
parentage. The following is a description given of him by Hasket Derby who became one of his pupils in 1859.

"He was an insignificant looking man rather below medium height, somewhat shabbily dressed in black, with sharp cut features, a large and suspiciously red nose; he was wearing a high stock of black satin and showed no collar. The latter in fact he never donned except on state occasions. His general appearance was so insignificant and ordinary that one of the class observed, on seeing him riding in his carriage, sitting up straight and perched on the extreme front edge of the seat, 'he looks as if he had been sent for a carriage by someone else and were bringing it home to him.' How his appearance belied him! He was the first of living operators, the most thorough and able of teachers, and a man of world-wide fame. Patients came to him from all over Europe. His 'Klinik' was thronged by patients of every clime." Such was the environment in which Fuchs commenced his training in ophthalmology and he always spoke with reverence and gratitude of what he had learnt from Arlt.

Arlt had a theory that acquired myopia was due to passive hyperaemia of the uveal tract from pressure of the external rectus and inferior oblique muscles upon the venae vorticosae during prolonged convergence. To test the truth of this theory, Fuchs undertook an investigation into the topography of the insertions of the extraocular muscles, of the vortex veins, and of their relation to one another. After careful measurements in a large number of eyes having different refractive conditions he arrived at the conclusion that the recti muscles could exert no pressure on the blood in the veins, but that the oblique muscles might do so. The worst of all positions in this respect being that in which both eyes are turned downwards and inwards. A position which might lead to obstruction of the venous outflow from the two outer venae vorticosae which take the blood from the posterior pole of the globe.

One of Fuchs's earliest contributions to the pathology of the eye, for which in subsequent years he did so much, was published in 1881, and consisted of an extensive and useful analysis of sarcoma of the uveal tract based on 259 cases collected from different sources. In the same year the University of Liège founded a Chair of Ophthalmology to which Fuchs, who was then thirty years of age, was appointed.

At the fourth International Congress of Hygiene, held at Geneva in 1882, the English Society for the Prevention of Blindness announced its intention of offering a prize of 2,000 francs for the best essay on the causes and prevention of blindness; the essay to be written in German, English, French, or Italian, and not previously published. In furtherance of the same object the International Society for the improvement of the condition of the Blind



HOFRAT PROFESSOR ERNST FUCHS.

offered a second prize of 1,000 francs and a third, with medal and diploma for the essay next in order of merit. Seven essays were received and the awards of the international jury were announced at the Fifth Congress of Hygiene held at the Hague in 1884. The first prize was awarded to Professor E. Fuchs, of Liège; the second to Dr. Wilbrand, of Hamburg; and the third to Dr. P. H. Mules, of Manchester.

Of Fuchs's essay the jury spoke as follows:—"It is an original work of great merit answering more completely than any of the others to the requirements laid down in the programme. Combining clinical experience with a complete knowledge of the literature of the subject, the author has considered all sides of the question with a thoroughness, exactness, and breadth of view which have struck all the members of the jury. Keeping constantly in mind the practical and philanthropic aim of the competition, and starting with a definition of blindness based on the social and economic position of the blind, the author succeeds in being complete and scientific, while he avoids superfluous statistics and considerations of pathology and therapeutics, which are outside his subject. His work presents a well-considered whole, of which each chapter may be profitably consulted by itself. The chief place is given throughout to the study of those preventive measures by which the number of incurably blind persons may be diminished. The jury feel it their duty to express the hope that this excellent work may soon be published, and if possible, translated into other languages, through the means of the English Society for Prevention of Blindness, or in some other way."

An English translation of this essay, made by Dr. Dudgeon, was published in 1886. The definition of blindness Fuchs gave, of which the jury so highly approved, was as follows: "Speaking scientifically blindness is that condition in which objective sensations of light are not received, but from the practical standpoint a man is blind if his eyesight is so far incurably impaired that he is unable to follow any calling which requires the use of the eyes." It is interesting to note how closely this corresponds to that arrived at for adults by a committee of the section of ophthalmology of the Royal Society of Medicine appointed in 1915 to consider the matter at the request of the Local Government Board's Departmental Committee on the Welfare of the Blind, and which has now been generally adopted. It reads, "the expression 'Blind' means too blind to perform work for which eyesight is essential."

It must have been a considerable source of satisfaction to Fuchs, considering all he had done to promote prophylactic measures against blindness, to be able, a year before his death, to quote the following figures showing a marked decrease in its amount. He wrote: "In 1899, there were on an average 8·7 blind in each

10,000 of population in the central and western European states; in Russia approximately 20, and in Egypt as many as 132. Just before the world war, figures for the central and western European states were about two-thirds of those in 1899—5·2 as against 8·7. The difference between then and now struck me forcibly in regard to Egypt. When I first visited there thirty-six years ago it was a common experience to see blind beggars asking for alms, one holding the other's hand, a chain of five of them led by one who still possessed some sight. And now one sees this no more. When I first visited the Arabic University of Al Azahr, in 1893, there were about 600 blind among the 4,000 students; at my last visit in 1926, I was told there were not more than 80."

The occurrence of crescents, due to choroidal atrophy at the outer margin of the optic disc in association with myopia, was recognised in the early days of ophthalmoscopic research. It was Fuchs, in 1882, who pointed out the necessity of differentiating between these acquired atrophic crescents, and congenital crescents due to a defect in the development of the choroid, occurring usually at the lower margin of the optic disc, which is small and oval horizontally. A condition commonly spoken of as Fuchs's coloboma.

In 1885 Fuchs returned to Vienna as successor to Professor Jaëger and in the same year published his classical article on the anatomy of the iris. Owing to the method of bleaching sections not having then been introduced, he was not able to give a full and accurate description of its posterior pigment epithelial layers and of its dilatator muscle fibres. In a later edition of his Textbook, however, he not only described the appearances shown in a bleached section, but also introduced a drawing depicting them.

The first edition of Fuchs's "Lehrbuch der Augenheilkunde" appeared in 1889. The purpose for which he originally wrote it was to supply those attending his lectures with the substance of his teaching in a permanent form, and thus relieve them of taking copious notes and of waste of time and energy in copying them out afterwards. Fuchs considered that the taking of notes distracted a student's attention from the object before him, more especially in clinical lectures. The book not only admirably fulfilled this, its original object, but as it passed through some twelve German editions and was translated into all the important European languages as well as Japanese and Chinese, it served to disseminate the author's thorough, clear and concise teaching throughout the ophthalmic world.

It was a happy idea to employ two different sized types in its construction, the essential portions of a subject being printed in large type, and the less important in small. By this means the book was adapted for the use of students studying for a pass degree, who subsequently intended to engage in general practice, and found

all that they would require in the matter in large type. Whilst those who studied ophthalmology, with the intention of devoting themselves specially to its practice, had matters of much additional interest placed before them in the small type.

The first English translation was ably carried out by Dr. Duane, of New York, from the second German Edition in 1892. A second English edition was published seven years later; and a third, likewise by Dr. Duane in 1908, from the eleventh, greatly revised and enlarged, German edition, containing 441 illustrations. These illustrations, both those of clinical and pathological conditions, most clearly indicate what they are intended to depict, being of considerable help in enabling the reader to follow the descriptions given in the text. Fuchs had but a poor opinion of photo-micrographs as a form of illustration and never employed them.

As already stated Arlt had made Vienna a great centre of ophthalmic teaching and treatment, and Fuchs not only maintained but still further enhanced its repute in both these directions. Patients who were dissatisfied with the treatment they were receiving or in whose case there was some difficulty in the diagnosis, journeyed to Vienna from all parts of the world to seek Fuchs's advice, which was always honest, straightforward and to the point; he never tried to magnify his capacities unduly. For there was never anything of the charlatan about Fuchs.

Shah Nasr-ed-din of Persia sent his favourite wife, who had become blind, to Vienna to consult Fuchs. It had been diagnosed that she had cataract and several other women suffering from cataract were allowed to accompany her. When Fuchs examined her he found she had absolute glaucoma and that nothing could be done for her. He was, however, able to restore the sight of the women who came with her. The Shah could never understand why it was that Fuchs could do so much for these women of low degree and yet nothing for his favourite consort.

The amount of clinical work which Fuchs's appointment as Professor of ophthalmology at Vienna involved is shown by a statement he made in 1897 in an article on "Retinitis Circinata." He there said that amongst 70,000 patients seen during seven and a half years in his clinic he had only observed eleven cases of this rare disease; one which he was first to differentiate from other forms of retinitis, and of which he gave such a full description of the clinical characteristics that little, if anything, has been added since.

Fuchs was a frequent visitor to England and seems to have enjoyed association with his British colleagues. When in London he stayed with his friend Dr. W. A. Brailey in Old Burlington Street, visited the operating theatre at Moorfields and gave encouragement and assistance to the younger men in their research work in its pathological laboratory.

He attended the meeting of the British Medical Association held in London in 1895, and read a paper at its Ophthalmic Section on "Erythropsia." His attention became attracted to the affection by having himself experienced it after wandering over the snowfields for some hours the previous year. He then proceeded to investigate it in himself and others in his usual systematic manner; climbing for the purpose to the top of a mountain 600 feet high near Vienna with a chess board, having black and white squares, and a campimeter. He came to the conclusion that erythropsia was a special phenomenon always presenting itself in the same way, and independent of the colour of the reacting light, provided that it be strong enough to produce dazzling. To explain it he inclined to the hypothesis that the visual purple having become completely bleached by exposure to light becomes visible as it regenerated in the dark.

Fuchs was a great walker and loved to spend his vacations in the mountains of the Tyrol or elsewhere, often accompanied by his friend and colleague, Professor Sattler, of Leipzig. On one occasion, when wandering in a wild part of Sicily, he was held up by brigands and had to part with his purse and watch.

Affections of the cornea was a subject in which throughout his life Fuchs took the keenest interest. In 1881 he wrote his description of the changes in opacity of the cornea in glaucoma, and one of his last publications was an article "On the Localization of Pathological Changes in the Cornea." A form of keratitis which will always be associated with his name is that which he termed "Keratitis Punctata Superficialis" of which he first gave a detailed account with drawings in 1889.

In 1902 Fuchs was invited to deliver the Bowman Lecture at the Ophthalmological Society of the United Kingdom and he chose for his subject, "Keratitis." In it he made a comparison of the corneal changes observed clinically with those found microscopically. He described the tendency which the epithelium has to level up all the inequalities of the corneal surface and keep it smooth; penetrating into clefts, and even sometimes, after extraction of cataract, extending down the wound into the anterior chamber, lining it, and causing glaucoma. He discussed desquamation and excitation of the epithelium, stated that he had no doubt as to the existence of neuropathic keratitis, and gave an account of the sequence of changes which occurred in what Arlt had termed "atheromatous ulceration," *i.e.*, ulceration in connection with old leucomata. Fuchs further briefly referred to the pathology of such rare conditions as Groenouw's nodular corneal opacity, ring abscess of the cornea, dystrophia epithelialis corneae, with all of which he dealt more fully in articles published in subsequent years.

Fuchs was an accomplished linguist speaking several different languages. His English delivery was so good that when he lectured

in that language it was a treat to listen to him. After the delivery of his Bowman lecture he was elected as one of the select list of Honorary Members of the Society.

Fuchs's love of travel led him to Ireland in 1909, and he attended the British Medical Association's meeting held that year in Belfast; accompanied by his wife he stayed whilst there with his old friend Dr. Nelson, who had been in his youth one of Garibaldi's red-shirt heroes. To the proceedings of the Ophthalmic Section he contributed a paper on "Malformations of the Cornea in cases of Inherited Syphilis," in which he pointed out the frequent coincidence of interstitial keratitis and vertical oval cornea. Out of 22 cases with vertically oval corneae which he collected in the course of a year, in 8 there was good evidence of inherited syphilis, in 8 others some indication of it, and 6 cases which exhibited no evidence of syphilis. This unusual form of the cornea he found influenced its curvature, inverse astigmatism being present in most of the cases to between 3 and 4 dioptres.

Fifty years ago it was the custom for a patient after an operation for extraction of cataract to be put to bed with both eyes tied up in a darkened room, and kept there for a week, the dressings being changed only by the light of a candle kept shaded at the foot of the bed. It was feared that the admission of light too soon to the eye would cause inflammation. The upgrowth of the science of bacteriology has changed all this, and in 1900 Fuchs did not hesitate to make an ophthalmoscopic examination of eyes within three days of a cataract or glaucoma operation, and found no evil results to arise therefrom. He further considered that the usual restriction as to moving about after cataract operations was unnecessary, and allowed his patients up out of bed the day following. As the result of his early ophthalmoscopic examination in these cases he found that detachment of the choroid was far from being an exceptional condition, as had generally been supposed, and instead of being difficult to diagnose could sometimes be seen by means of focal illumination. In the paper which he wrote on the subject in 1901, he estimated the relative frequency of its occurrence after different operations, the conditions which predispose to it, the time after an operation at which it most frequently occurs, and discussed its pathology.

Fuchs's keen capacity of observation and accurate methods of investigation are well shown in two papers which he published in 1904 and 1905, which form important contributions to our knowledge of inflammations of the uveal tract which follow perforating injuries of the eyeball. He pointed out the difference between exudative uveitis, which he called "Endophthalmitis," and the granulomatous infiltration of the tissue which is associated with sympathetic inflammation.

In endophthalmitis the exudate consists of lymphocytes and polynuclear leucocytes, the inner layers of the ciliary body and the retina becoming involved, and the choroid only later when the retina is so disorganised as to cease to shield it. In sympathetic ophthalmitis on the other hand the proliferation consists of a diffuse infiltration with round cells, accompanied by proliferation of endothelial cells and giant cells; the stroma of the uveal tract being first involved, either uniformly throughout or in the form of nodes. With these essential differences in view, Fuchs proceeded to examine sections of 181 eyes removed after injuries for the fear of sympathetic disease during a period of twenty years. Of these he selected by microscopical study, and without knowing the clinical histories the sections of 24 eyes showing the granulomatous proliferative form of uveitis; subsequent reference to his notes showed that the fellow eye in all these cases except one became sympathetically inflamed. In the one exception there was a history of sympathetic irritation, but in 16 other cases of sympathetic irritation there were no signs whatever of proliferative uveitis in the enucleated eyes. Though exudative endophthalmitis may be present together with proliferative granulomatous uveitis, it is only in the presence of the latter that the fellow eye is likely to become involved.

At the International Medical Congress held in London in 1913 Fuchs was invited to be one of the openers in the Ophthalmic Section of the discussion on "The Pathogenesis of Chronic Uveitis, excluding Syphilitic, Tuberculous and Sympathetic Cases." He dealt mainly with the pathology of the condition, describing the characteristics of the cells in the exudate and the different forms of deposit on the posterior surface of the cornea. He classified cases clinically into five groups according to their degrees of severity.

In 1908 Fuchs undertook a detailed investigation of cases of tumours of the ciliary epithelium; not only of those which he had had under his own observation, but also of cases published by other observers to whom in many cases he applied for and obtained sections which enabled him to inspect them personally. As the outcome of this work he suggested the provisional classification of such tumours as follows:—

I. Innocent tumours (so-called adenomata).

II. Malignant tumours.

1. Having the structure of the embryonic retina (Diktyoma).
2. Whose structure does not represent the whole retina but at most the simple ciliary epithelium. These consist of
   - *a*. Cellular membranes with a single layer of nuclei, combined with cellular tubes.
   - *b*. Cellular tubes alone, or
   - *c*. Cells grouped in an irregular fashion with only a few tubular formations in the youngest parts.

Usually when a man reaches the age of 60, his capacity and energy for conducting original investigations commences to wane. It was not so with Professor Fuchs. The compulsory age for retirement from ophthalmic hospital appointments in this country is usually 60. Fortunately for ophthalmology the legal age for retirement from the chair of ophthalmology in Vienna is not until 70. Up to that age Fuchs remained full of vigour, not only carrying on his routine duties, but also attending and taking part in discussions at the annual meetings of the Heidelberg Ophthalmological Congress, which he always preferred to the larger International Congresses, and continuing his output of valuable clinical and pathological researches until the outbreak of the great war.

In 1911, he visited the United States of America and at its Ophthalmological Society delighted his audience with a paper on "The field of vision in Tabetic Atrophy of the Optic Nerve." In it he maintained that the proportion of cases in which a central scotoma was present had been considerably underestimated, and that it was one of the results of the tabes and not, as had been suggested, a complication due to a simultaneous syphilitic retrobulbar neuritis. It was, however, he said, still uncertain whether tabetic changes in the nerve were purely degenerative from the beginning or whether a slight form of inflammation preceded the atrophy.

Most of Fuchs's papers were published in von Graefe's *Archiv fur Ophthalmologie;* in 1913 he published one in French in the *Annales d'Oculistique* on "Opacity of the Lens after Corneal Suppuration," in which he suggested an explanation of the pathogenesis of anterior polar cataracts, some of which occur in connection with perforating ulcers of the cornea; some with ulcers which do not perforate; and some, which are congenital, without any sign of corneal inflammation. He discounted the hypothesis of a previous writer, that the primary change was a shrinkage of the lens fibres at the seat of the opacity, by attributing such shrinkage to the hardening reagent. He described the first change as a rarefaction and destruction of some of the epithelial cells of the anterior capsule, followed by proliferation of neighbouring cells. The primary destruction, in cases of corneal suppuration, he attributed to toxins in the aqueous humour; but did not explain why such toxins should affect the cells lining the lens capsule and leave unaffected those lining Descemet's membrane. Where capsular cataracts developed as the result of an anterior synechia of the membrane, he attributed the proliferation of its lining cells to a negative pressure produced beneath the capsule, apart from any toxic influence.

Some difference of opinion existing as to whether a rupture of the sclerotic proceeded from without inwards or from within outwards; Fuchs, in two articles published in 1911 and 1913, brought to bear

the wealth of his clinical and pathological material in elucidating the point. In the first paper he clearly showed that ruptures began at the ligamentum, passing to Schlemm's canal and then outwards towards the surface. In the second paper he described a special variety of rupture of a smaller type, confined to the limbus, beginning at Schlemm's canal and extending straight forwards instead of diagonally outwards; in which the conjunctiva was always torn and where generally there was a prolapse of iris. He also referred to another class of incomplete rupture in which the conjunctiva and outer layers of the sclera remained intact; the area involved, later becoming ectatic, and the pupil displaced upwards.

As the outcome of his many years of hard work Fuchs was before the out-break of the great war in affluent circumstances, and delighted in hospitality, gathering around him at his dinner table visitors to Vienna from all parts of the world. At the conclusion of the war, due to the rapid depreciation in the value of the krone, he became considerably impoverished. At the commencement of his eighth decade he met his altered circumstances in the most courageous fashion, and accepted an offer gracefully and opportunely made in 1921, by his friends and admirers in America to conduct a lecturing tour in the United States on the pathology of the eye. His love of travel subsequently led him to visit Spain, Egypt and Asia Minor.

In 1929 he motored across Europe, with his old friend Dr. Barkan, some five years his senior, to attend the International Ophthalmological Congress in Amsterdam. Here a great welcome awaited him. To a group of American ophthalmologists attending the congress had occurred the idea of organising a luncheon in honour of this Grand Old Man of Ophthalmology. A large gathering of his friends, pupils and admirers from all countries assembled, with Dr. Suker in the Chair, and when Hofrat Professor Fuchs's health was proposed the toast was received with resounding applause. The opportunity was also taken of presenting to him the Leslie Dana Medal, instituted as an award for work conducive to the prevention of blindness. Fuchs's reply to this enthusiastic reception was characterised by his usual tact and modesty.

From Holland he proceeded again to America to attend and take part in the dedication ceremony of the Wilmer Institute of Ophthalmology in Baltimore. Afterwards, before returning home, he extended his travels to Mexico. Most truly may it be said that Fuchs never allowed grass to grow under his feet.

As a man, Fuchs possessed a striking appearance; well above the average height, he had the slight stoop of a student. A flowing beard made his naturally large head appear even more massive, but his most conspicuous features were his observant, watchful grey eyes. The expression of his face was always calm and unruffled;

indeed it would be difficult to have imagined him as angry or impatient.

Fuchs leaves behind him not only a store of most valuable scientific work, but also the example of a fine character and of one who hungered and thirsted after truth.

E.T.C.

RETINITIS NEPHRITICA OR ALBUMINURICA

BY

N. PINES

LONDON

"*Ceterum censeo ophthalmoscopum quotidie usendum esse.*"

THIS paper is the third and last part of one chain of clinical investigation. All the work is the outcome of general practice where the ophthalmoscope was used merely as a means of thorough clinical examination, limited by the comparatively simple resources of routine surgery-work and by the necessity of regarding my retinal findings as a part of the clinical whole. This work is, therefore, as much an essay in general medicine as in ophthalmology and its chief value, if any, lies in demonstrating the importance of an *early* ophthalmoscopic examination. As an instance it is sufficient to point out the books of Fisher on oedema and McDonagh on the nature of the disease where the ophthalmoscopic examination is wholly omitted. The same applies to many of the cases quoted in D. Russell's remarkable book on Classification of Bright's Disease.

In the special ophthalmological literature up to the present day there still exists a very wide divergence of views on retinitis albuminurica. Putting aside purely pathological or anatomical treatises it will be sufficient to quote the opinions of only a few recognized authorities. In Fuchs' well-known text-book there is no difference between the eleventh and seventeenth editions; in both of them this learned writer considers that "the severity of retinitis albuminurica bears no fixed proportion to the intensity of the kidney disease nor to the amount of albumin in the urine ..... The like is true of the subsequent course; the retinitis may improve, while the kidney lesion grows worse and *vice versa* ....." Fuchs believes that *every* form of nephritis, which is accompanied by albuminuria, may be complicated by retinitis albuminurica. Fox, in his text-book, expresses a similar view. Leber, in the Graefe-Saemisch handbook, holds a different view. On page 817, Vol. VII,

he writes that the retinal changes can regress if the basal pathological process in the kidney is healed or improved; in favourable cases even highly advanced retinal changes can regress completely. But though Leber admits the dependence of retinitis on the kidney, he also holds that retinitis albuminurica apoplectica (haemorrhagic retinitis) is most probably connected primarily not with the kidney, but with the vascular system (p. 825). Poulard expresses the opinion that the appearance and prognosis of retinitis is closely connected with the impairment of the function of the kidney as shewn by bio-chemical tests. Duke-Elder believes that most probably the aetiology of retinitis albuminurica, diabetica and sclerotica is the same for all of them. For Fox retinitis albuminurica is essentially arterio-sclerosis of the smaller and, when advanced, of the larger retinal vessels. Lachmann (*Medizin. Welt.*, Vol. XXXVIII, 1929) thinks that albuminuric retinitis is often the first warning sign of renal disease, but the latter need not necessarily be severe.

My own conclusions are reached on purely clinical observations, only one of my cases having been verified by an operation and a post-mortem examination. This alone makes my exploration of the subject necessarily incomplete, but it still affords an opportunity of discussing many general pathological problems.

The regulation of urinary secretion, roughly speaking, depends on:—

(1) The chemical composition of the blood which is brought to the vascular tree of the nephron. This alone can produce locally an arterial spasm.

(2) The height of the blood pressure and the velocity of the blood circulation, with all their causative factors (central and peripheral heart, elimination of CO_2 from the blood, etc.)

(3) The central nervous system through vasomotor, reflex and psychic stimuli; special centres concerned with renal secretion (Hall).

(4) The endocrine glands—the most obscure branch of medical knowledge; but diabetes mellitus is a good example of this factor.

(5) Local metabolism of the kidney cells, be it increased acidity, alkalinity or otherwise. This is probably the most important factor of all, but extremely difficult to determine in our present state of knowledge.

Nephritis

In disease the different parts of the kidney may be attacked—in most cases they are all more or less involved—and this makes the classification and differentiation of the various forms of nephritis

extremely difficult. The old classification based chiefly upon the morphological and microscopical aspects of the organ, as seen in the post-mortem room, did not pay sufficient attention to clinical observations, and the attempt to correlate it with the findings of modern bio-chemistry failed. It is more important for the clinician to realize to what extent the efficiency of the kidney is damaged and what part of the whole clinical picture should be attributed to it, than to try and guess what may be the histological state of the organ. When a decapsulation was being performed on a kidney of one of my patients, the surgeon asked the two physicians present the size of the kidney. " Contracted, most probably " one replied; "fairly normal" was the opinion of the other. " It is enlarged, gentlemen!" answered the surgeon, delivering the kidney from the wound and starting the decapsulation. And yet both physicians knew perfectly well all the clinical features of this exceptional case.

" Nephrosis " is the name given to the disease of the tubular or proper kidney cells only. In this country some authorities object to this term; in their opinion nephrosis is simply a form of parenchymatous nephritis. On the Continent and in the United States of America this name is commonly used as distinct from nephritis. Moorhead prefers the term " toxic " kidney to the term " nephrosis " and he is correct from the aetiological point of view. Most authorities state definitely that the cause of this disease is extra-renal; that it does not originate in the kidney, but that some toxic factor attacks all the tissues of the body and that of the kidney as well. (See for instance, " Nephrosis in Children," by Gladys Boyd). Clinically nephrosis is characterized by a marked tendency of the tissues to become oedematous, the cardiac activity usually being unimpaired, the blood pressure normal but the urine shows enormous quantities of albumin, waxy casts, epithelial elements, lipoids or fats and the absence of erythrocytes; the specific gravity is high; and there is oliguria, with diminished chloride content; the alkaline reserve of the blood is very high. It is a disease of the whole organism in which, clinically, the kidneys play an entirely subordinate rôle. It is important to note for the purpose of this paper that in this rare disease three phenomena are never present—erythrocytes in the urine, arterial hypertension and retinitis. There is no disturbance in the nitrogenous metabolism and the oxidation process in the tissues is normal as witnessed by the normal blood urea. The basal metabolism is frequently reduced. In the initial stage ascites and hydrothorax are very marked, but they disappear later. In Schlayer's opinion, oedema following an infectious disease, associated with a haemorrhagic exacerbation in the urine, vascular changes in the fundi oculorum, arterial hypertension with the urea excretion diminished—all these are in favour

of a nephrosis with a "nephrotic" character or chronic glomerulo-nephritis. Saxl, of Vienna, very definitely states that in nephrosis there is no hypertonia and that the latter appears only with the vascular lesions of nephritis or of nephrosclerosis; as a matter of fact a contracted kidney can develop from pure nephrosis. *There is no retinitis so long as the kidney is not contracted,* and no tendency to the retention of nitrogen (azotaemic uraemia). Neither are eclamptic attacks (convulsive uraemia) observed because the blood pressure is normal. Histologically the kidney usually shews intact glomeruli and severe fatty, albuminoid or amyloid degeneration of the tubular cells, which contain a lipoid, doubly refracting substance. The kidney is enlarged, soft and of a white or yellow appearance. It corresponds to what is called the large white kidney, or to the old name of parenchymatous nephritis. Some authors think that the excretion of urine may be insufficient and thus is caused an occasional attack of diarrhoea (similar to that of amyloidosis), but Schlayer states that usually an increased quantity of urine is passed by the nephrotic kidney. The prognosis is, as a rule, not very unfavourable, the disease generally lasting for years and the chief danger being a secondary infection, because the general defensive apparatus of the body is impaired. Recovery is possible. In Munk's opinion, the prognosis is usually favourable if there are no signs of glomerulo-nephritis (arterial hypertension, retention of nitrogen, erythrocytes in the urine). These are the characteristic signs of a pure nephrosis. But very often " nephrotic " appearances accompany other diseases and then the diagnosis becomes difficult. The albuminuria of fever means an albuminoid degeneration of the tubules—is it a nephrosis or a mild form of nephritis? The same applies to syphilis, especially in the secondary stage. Intoxications may lead to nephrosis and to chronic glomerulo-nephritis. The case of a diabetic patient is ever more striking—is albuminuria here an early sign of a commencing chronic glomerulo-nephritis (as is usually thought) or of nephro-sclerosis or is it only nephrosis? Estimation of the blood sugar does not help. Even the sphygmomanometer may not be very useful, as diabetes *per se* may be accompanied by vascular disease and, moreover, if the myocardium be impaired, it cannot produce a very high systolic pressure. Here again the ophthalmoscope, if used carefully, frequently and in a proper manner, may help in the diagnosis, as pointed out in my previous papers. The same applies to the albuminuria of pregnancy.

Osler points out the important fact that the glomeruli and tubules may temporarily substitute one another in their action to a certain degree, but the tubular cells, not used to the " high speed and pressure " of the glomeruli, will suffer considerably. The more so because the tubular cells are supplied by the afferent artery

of the glomerulus and any toxins affecting the latter will affect the former also. If we add to this a possible damage from the toxins that pass through the glomeruli and travel down the tubules we shall readily understand how early " nephrosis " may accompany the other forms of nephritis. Such may be the case in glomerulo-nephritis. A patient may be seen with hypertension, haematuria, retinitis, a tendency to uraemia—all signs of glomerulo-nephritis. But his urine may be saturated with protein and his body may be markedly oedematous. If the Fates are benevolent, the haematuria will disappear, as will also the tendency to uraemia. The blood pressure may become nearly normal, but the retinal changes, even if regressed, will remain as a silent and permanent witness of a vicious vascular past. The urine may remain very dark in colour and contain large quantities of protein and lipoid, and oedema of the body will persist—the nephrosis has not disappeared. Even pure vascular disease of the kidney vessels, such as arterio-sclerosis and atheroma, according to some writers may lead to nephrosis by interfering with the nutrition of the tubular epithelium.

It will take us far away from the theme of this paper if we describe or discuss the many other features of nephrosis; but some important points are already clear. Fisher considers that every albuminuria means a loss of kidney substance. The patient would soon succumb, as the kidneys are not very voluminous organs, if later on the albumin were not supplied by the damaged glomeruli and ruptured kidney vessels. But the latter source means the appearance of erythrocytes in the urine and their absence is characteristic of nephrosis. The course of nephrosis is a very prolonged one—*eo ipse* the albumin is not the result of a loss of kidney substance. The same objection—absence of erythrocytes in the urine of a nephrotic— applies to the filtration theory of urine, which always presupposes damage to the glomeruli in every case of albuminuria. *Albumin in the urine may be present in an enormous amount as the result of a pathological metabolism of the tubular cells, produced by an extrarenal cause, without occasioning death of these cells, without arterial hypertension and without producing retinitis albuminurica.*

In acute nephritis all the elements of the kidney are most probably involved, but any one of them may be more seriously affected than the others. In haemorrhagic nephritis we have a more benign form of the disease; it commences without oedema, which if present, only occurs late; arterial hypertension and hypertrophy of the heart are seldom observed; retinitis albuminurica is extremely rare, but if it be present the prognosis is very serious indeed. Haematuria remains the chief symptom, and transition to chronic glomerulo-nephritis seldom occurs. In the majority of

cases the cause of the disease is in the tonsils, nasal accessory sinuses, etc. The second form—acute oedematous nephritis is much more serious. The heart is usually affected and the blood pressure increased. The prognosis and treatment depend on careful clinical observation, paying special attention to the heart, blood pressure and fundus oculi (Schlayer). Retinitis albuminurica may be seen and, if present, makes the prognosis very gloomy. Isaac thinks that the dyspnoea, oedema, increase of blood pressure, scanty diuresis and the inclination to convulsions are due more to the disturbance of the circulation than to the kidney itself. Eppinger thinks that hypertonia and oedema (especially hydrothorax, as demonstrated by X-rays) may occur even before albuminuria is evident. An increase of blood pressure was at one time regarded as almost certain evidence of old-standing nephritis. It is now, however, universally recognized that even in primary acute attacks of every variety of nephritis the blood pressure may rise rapidly (Moorhead). The reader will see later that this point is still not settled in regard to the kidney of pregnancy. It is important for us to note that in acute nephritis retinitis albuminurica is fortunately very rare and is always preceded by arterial hypertension and other signs of serious damage to the glomeruli and to the general cardio-vascular system.

In the great majority of cases acute nephritis subsides and the kidney recovers, but in other cases the latter becomes chronically affected, the disease spreading slowly and insidiously, involving one nephron after another, crippling them or putting them out of action. Volhard ingeniously explained the difference by the degree of impairment of the blood supply to the glomeruli. And may I suggest that one of the causes may be the same collapse of the artery, a result of an oedematous perivascularis as seen on the retina? If the tubular elements are chiefly involved, the patient will suffer from chronic parenchymatous nephritis with considerable albuminuria and normal or moderately raised blood pressure. The heart will be involved and will suffer from myocarditis, or if hyperpiesis be present, from hypertrophy and enlargement. Retinal changes may be present or absent; if present, this will be in a patient with hyperpiesis and the prognosis will be bad. The blood urea may be normal for a long time and become increased only later, in the terminal stage. Oedema of the tissues is usually definitely marked and the excretion of sodium chloride by the kidney may be impaired. This form of nephritis is the "chlorure" or " hydraemic " type of Widal and other French authors. If the glomerular involvement is the greater we have the " azotaemic " type of Bright's disease, with traces of albuminuria, no oedema of the tissues, elevated blood pressures, hypertrophy of the heart and retinal changes. The blood urea is raised, and dyes and, of

course, urea are badly excreted by the kidneys. In a very interesting paper on chronic nephritis in childhood (*Brit. Med. Jl.*, Aug., 1928), Spence stated that retinitis occurred in cases with high blood pressure. Very rarely does one see such a case of primary arterio-sclerosis with secondary contraction of the kidney as occurred in a child of ten with a systolic blood pressure of 220 mm.; renal efficiency tests were normal, but so-called uraemic symptoms occurred, such as transient amaurosis and monoplegia of the left arm, which, in the presence of albuminuria and retinitis, were regarded as uraemic, although they were more likely due to a cerebral vascular disturbance. In all the cases of chronic interstitial nephritis with normal or low blood pressure retinitis was absent, and Spence concluded that when it did occur retinitis was a manifestation of hypertension or vascular disease. Davies saw many case of *uraemia* in children : *even when very ill these children maintained an alert mental condition.* The blood pressure was not raised and absence of eye-signs was the rule .... Remarkably high blood urea, 300 mg. per 100 c.c., was noted.

The kidney may be the seat of a vascular disease that has attacked all the organs of the body, *e.g.*, the angio-fibrosis of Sutton, the involutionary arterio-sclerosis of Allbutt, etc. It will also be involved in essential hyperpiesis, called " vascular disease " by many authors. Involutionary arterio-sclerosis implies only a limitation of function that occurs *pari passu* with the same process throughout the body; the kidney is, therefore, quite capable of serving the organism's needs and no danger to life exists from that circumstance. The bio-chemical tests are usually normal. The kidney changes are only slightly progressive and in view of the reduced metabolism the patient does not reach the stage of renal insufficiency. In the benign form of essential hyperpiesis the renal vessels are also attacked, and I agree with Lewandowsky and others when they suggest that a permanent systolic blood pressure of over 200 mm. means a wide-spread sclerosis of the kidney (nephro-sclerosis) or ischaemic nephritis (D. Russell). The glomeruli may be moderately involved or be only partly out of action and a sufficient number remains to give satisfactory bio-chemical tests and enable one to carry on for many years. The sclerosis may involve chiefly the afferent arteries and the glomeruli will suffer secondarily and not primarily. In years, if this process advances and an insufficient portion of the kidney is left for action, the patient will reach the stage of renal insufficiency—" malignant nephrosclerosis." Essential hyperpiesis may be malignant from the onset and then it means a rapid progress of the disease; the expectation of life may be reduced to five or six years, and in the end the *clinical* picture will be identical with the terminal stage of true interstitial nephritis. In spite of

relatively good results from bio-chemical renal tests we have to watch the cardio-vascular system very carefully in both forms. One cannot agree with the opinion of D. Russell, that primary ischaemic nephritis never interferes with renal function. It may lead to albuminuric retinitis and it was so in her own case No. 28.

In " uraemic conditions " Volhard and many other authorities distinguish two different pathological agencies—vascular disease and renal insufficiency. If both kidneys are excised from an animal, the latter preserves until the end mental alertness, good vision and complete consciousness. The same phenomena have been mentioned previously when describing nephritis in children. The animal dies from gradual wasting and weakness, vomiting, etc. Experimental injections into animals of large quantities of urea failed to produce a state of uraemia, and many authors believe that urea *per se* is most probably a quite innocuous substance. Generally speaking not one of the chemical substances that are employed clinically for renal efficiency tests has been proved to be the cause of uraemia. The convulsions, the cerebral disturbance, the diseased heart are all, in Volhard's opinion, the result of vascular disease. Fisher goes much further than this. On page 883 he writes: " It is not the kidney disease that leads to the high blood pressure, cardiac hypertrophy, etc., but it is the vascular disease, which when it happens to affect the kidney gives rise to albumin and casts." The high blood pressure, cardiac hypertrophy, etc., so frequently observed with this type of chronic interstitial nephritis are not the consequence of the kidney disease, but expression of the vascular disease here held responsible for the kidney lesion ..... Blood vessel disease, high blood pressure and cardiac hypertrophy are not secondary to loss of kidney function " (p. 24). The first sign of impaired renal function is given by a simultaneous rise in the urea and sodium chloride in the blood as well as of the systolic blood pressure, and it is only when the renal lesion has become more or less pronounced that it is detected by examination of the urine and general condition. (Brasiello).

Blacklock and MacDonald in their paper on blackwater fever conclude that in this disease a state of anoxaemia is present, which causes an increase of sarcolactic acid either locally or generally. " We produce evidence from experiments, both *in vitro* and *in vivo*, that sarcolactic acid is haemolytic ..... to whole human blood ..... The sarcolactic acid is the causal agent in the production of the haemolysis in blackwater fever and these haemoglobinurias." Moreover it is quite a common occurrence to find albuminuria, casts and even occasional erythrocytes in the urine after strenuous muscular exercise. The brain in the living animal produces lactic acid very rapidly from some precursor, so rapidly that, when all possible haste is made in killing, excising, and fixing the tissue

only a very little extra lactic acid can subsequently be formed *in vitro*, unless glucose be supplied artificially." (Holmes).

McLean says: "The increased blood pressure of the acute disease is almost invariably present when oedema is well marked and it quickly returns to a normal or even an unusual low level with subsidence of the oedema" (p. 187). "Hydraemia ..... is closely connected with the rise of blood pressure." Although in the account of his cases McLean does not give a description of their fundi (this, in my opinion, considerably diminishes their clinical value), he says on page 27: "There is a constant amount of evidence pointing to the conclusion that these eye changes are connected with the raised blood pressure." "Speaking very broadly, it may be taken for granted that in chronic renal disease, the more serious the renal damage, the higher the blood pressure and the greater the cardio-vascular involvement" (p. 75). "Arterio-sclerosis in a patient with comparatively active kidneys is often not so hopeless as it would be if the kidney were badly involved, and it is now generally admitted by those who have carefully studied the subject that arterio-sclerosis may sometimes be present without the kidneys being much, if at all, involved." A perfectly true sentence, which is difficult to understand, because McLean uses the vague term of arterio-sclerosis. If he means "involutionary arterio-sclerosis" or "athero-sclerosis," he is correct; if "essential hyperpiesis" he is wrong. "Extensive renal disease may exist with little or no increase of blood pressure ..... arterio-sclerosis may be present without much renal involvement."

Elwyn in his "Nephritis" states that in the senile kidney the renal artery is sclerotic; in hyperpiesis the primary cause of the changes in the kidney is the sclerosis of the arterioles of the kidney. In nephrosis the tubules are involved, but the blood pressure is not raised and no eye changes are met with. Diminution of kidney efficiency is usually combined with a rise of blood pressure. *The hypertrophy of the heart is the result of the raised blood pressure.* Osler describes the microscopic findings in chronic interstitial nephritis as a general increase in the fibrous tissue, thickening of the vessel walls, destruction of the glomeruli and more or less marked degeneration of the renal epithelium. "The glomerular lesion, however, is not necessarily primary. The more correct conception is to regard the process as from the first more or less diffuse, and to look upon a change in one part as necessarily leading to a change in another, often in the manner of a vicious circle." (p. 896). "An endarteritis or even mesarteritis can usually be made out ..... the smaller intertubular vessels are specially involved and are often entirely obliterated ..... The sclerosis is sometimes patchy, even in the individual artery, one portion of the circum-

ference shewing an extreme lesion, while the remainder of the ring is nearly normal." " Exceptionally typical contracted kidney has been found unaccompanied by arterio-sclerosis, or cardiac hypertrophy. Roth reports six such cases. Death was generally from uraemia. In most of the cases blood pressure had been low" (p. 899). He does not mention the importance of the diastolic blood pressure. The secretion of water by the kidneys depends in his opinion on the rapidity of the flow of blood through the renal vessels; this may or may not be influenced by a rise or fall of the blood pressure. The local activity of the vasomotors in the kidney vessels is much more important. " The tubular cell is prompt to suffer from the effects of even transitory toxicity of the blood and lymph (p. 772) ..... The toxicity, which is sufficient to kill a tubule cell, only irritates the interstitial tissue—the cell perishes and its work is thrown on to the other cells; the interstitial tissue grows under the double influence of the toxin and the empty space left by the cell."

Stevens divides nephritis into three groups. The first—tubular nephritis; the second—inflammation and degeneration in and around the Malpighian bodies; and the third—increase of the fibrous connective tissue around the glomeruli and between the tubules and sclerosis of the small blood vessels (renal sclerosis). " Renal sclerosis is always an end stage of some earlier process affecting the kidneys." " In chronic glomerular nephritis the blood pressure is usually almost invariably increased, a systolic pressure of 200 to 250 (Osler's figure starts from 170) and a diastolic figure of 120 to 160 being not unusual."

Lyon recognizes four types of nephritis:

(1) Simple albuminuric nephritis without any oedema, cardio-vascular changes or " uraemic " symptoms. The albuminuria is not influenced by rest or dieting. The prognosis is favourable because the evolution of the disease is very prolonged.

(2) Chronic hydraemic nephritis. The urine is scanty and concentrated. Oedema of the tissues is present. Sodium chloride is not excreted by the kidney, but is retained in the tissues. The water is retained by the tissues to dissolve this sodium chloride. This is the cause of the oedema. This form of nephritis is apt to be complicated by nitrogen retention.

(3) Uraemigenic nephritis. The blood urea is raised. "L'azotémie se traduit par les troubles nerveux, surtout cérébraux, et par les troubles gastro-intestinaux."

The nervous features include fatigue, torpor, headache and retinitis albuminurica, the gastro-intestinal disorders, intense anaemia and loss of flesh.

(4) Nephritis with arterial hypertension, the blood pressure being extremely raised (he mentions only the systolic, omitting the diastolic) and cardiac hypertrophy. Albuminuria is only slight or absent. The blood urea is not raised. There is no retention of chlorides. The patients are subject to haemorrhages including epistaxis and retinal haemorrhages.

From this imperfect review of the observations of a few modern authorities, necessarily incomplete because it is adapted only to the subject matter of this paper, the reader will realize that there is no uniformity of opinion about the physiology and pathology of the kidney and, moreover, that the opinions on some vital points are contradictory. See also page 14 in H. French's book. So far as retinitis albuminurica is concerned, I believe that an analysis of my cases will help to some extent to bring more order out of the present chaos, and it may probably throw light upon some doubtful points of general significance. Further work is needed, in D. Russell's opinion, to determine what other clinical criteria can help to separate primary ischaemic nephritis from the latent stage of nephritis repens. And it is my deep conviction that the ophthalmoscope may be the ultimate judge in this difficult problem.

Retinal Oedema

The general view is that oedema of the tissues is of extra-renal origin. Such, for instance, is the view of McLean and of Bennett. It is held that toxins damage the metabolism of the cells, causing water to be retained by the tissues. Our ignorance of the functions of the so-called interstitial tissue prevents us from understanding why the fluid collects in the different parts of the body in certain varieties of oedema. We still do not possess a theory that would solve all the difficulties of this thorny problem. It seems to me that Fisher comes nearer than anybody else to the correct solution. By very ingenious experiments he excluded the influence of the blood circulation. For instance, he placed a ligature over the knee of a frog and tied it sufficiently to stop the arterial and venous circulation. The other leg was left intact. The frog was put into distilled water sufficient to cover both legs and the ligatured leg with time swelled enormously, but the other did not. The explanation according to Fisher lies in the fact that the colloids become acid very quickly after their supply of oxygen is cut off. Being more acid the colloids absorb water and become oedematous. If the frog is put into a dry vessel the ligatured leg does not swell; it mummifies with time. The same explanation applies to the swelling of dead animals or of individual organs when separated from the body. Fisher thinks that the cause of the acidity is CO_2, which is the result of cell metabolism. At its arterial end the cell

receives nutritious material and oxygen and the formed CO_2 is washed away at the venous end of the cell by the lymph or blood. Modern investigators tacitly admit the truth of this theory, but they add other acids, especially lactic acid, to the CO_2. " In pneumonia there are two things wrong with the patient, a general toxaemia and the local changes in the lungs. Usually the toxaemia is the cause of death, and the oxygen seems to do good by neutralizing the effects of the toxaemia as well as possibly by its action on the micro-organisms. Toxaemia kills by producing anoxaemia, with reduction of the alkalinity of the blood, and the condition of acidosis is produced ..... The kidney and liver, the organs apparently responsible for maintaining the proper alkalinity of the blood, may both be upset." (Kirk, *Brit. Med. Jl.*, Aug., 1928). In my opinion, the above theory of Fisher taken along with what we know from Pal about vascular spasm is the best explanation of what is seen in the retina when there is an interference with its oxygen supply. All the brain tissue and, therefore, the retina " uses a considerable amount of oxygen and its metabolism must be of considerable magnitude. Lactic acid is formed by the brain tissue and this so rapidly that it is extremely difficult to eliminate it when preparing tissue for microscopical examination." (Eric Holmes, *Brit. Med. Jl.*, Sept., 1928). Menzies ascribes to anoxaemia of toxic origin the explanation of many problems of psychiatric pathology. This by itself is a sufficient explanation of what we see ophthalmoscopically in the living retina. Fisher entirely ignores the backward pressure of the blood circulation and, in my opinion, he is wrong in this minor detail.

As an illustration of the onset of retinal oedema as the result of local metabolic disturbances, cases of embolism or of sudden thrombosis of the central retinal artery or one of its branches, serve well. In such cases larger arteries can still be seen as very narrow lines; the smaller ones disappear entirely. Later, within a few days or weeks, the arteries can again be seen and the retina is once more transparent. I had an opportunity of examining a patient a few hours after he had suddenly become hemianopic in the right eye as a result of thrombosis of the upper branch of the retinal artery. I knew the appearance of his fundus before. I could see all his arteries, even the smaller ones, but they were covered with white sheaths of oedematous perivascular membrane. He did not present the well-known areas of retinal exudation, so characteristic of retinitis albuminurica; the retina was uniformly oedematous, but the vessels were sharply differentiated as swollen and whitish lines. The blood stream in them, although diminished, was not completely interrupted. Many of the arteries were free, and a few of them well covered in certain parts. That it was a transitory interference with the arterial circulation was evident

from the fact that I could see the blood stream in the artery immediately after the attack and later on as well. It was sufficient to cause irreparable damage to the function of the cells of the retina, as witnessed by permanent blindness, but not powerful enough to cause severe degeneration with scarring. The retina was absolutely transparent three months later. Sometimes, after an exudation or a haemorrhage disappears, an area of atrophy is left as witnessed by a whitish colour. But the oedema of the retina seen in such cases of vascular disturbance was not confined to a localized area, nor did it cover the retinal vessels; the latter were concealed only by their oedematous perivascular sheaths. As the blood supply was interrupted or considerably diminished, it is difficult to explain the resulting oedema otherwise than by increased acidosis of the retinal cells with retention by them of water and possibly different salts, and, consequently, with impediment of the back-flow of lymph in the perivascular channels.

In contrast to the exudates associated with oedema that are seen in albuminuric retinitis there is the fundus of retinal sclerosis. Here the characteristic feature is the absence of noticeable oedema. Apart from haemorrhages one often sees a small area of sharply defined bluish-white exudation. It is usually situated not very far from a vessel, sometimes even adjoining it. The colour differs from the " fat " colour of an albuminuric exudation. The surface is always flat—it seems to be on the same level as the retina; the albuminuric type sometimes looks prominent or elevated. The most important feature is that its edge is sharply limited; there is no oedema, no gradual transition into the surrounding retina as is the case with an albuminuric exudation. A vessel crossing such a sclerotic fundus will be clearly seen, sharply outlined, not oedematous and not covered by a veil. Sometimes in a very advanced exudation which leads to a scar in the retina the vessel, especially a vein, will be narrowed at this place, but will still be clearly seen—in my opinion, the best proof that it is the perivascular channel which is involved in the scarring originating from below the vessel. Careful attention must be paid to local perivasculitis, as I described elsewhere. (It may be advisable to mention here that even a normal retinal artery may shew a slight constriction of the lumen in some parts, especially on the disc. This phenomenon is not described till now and I would warn against considering those physiological local constrictions of the vessels as being pathological. In a few cases I saw one of the large arteries become gradually narrower from the periphery of the disc towards its centre exactly in the same way, as veins show this appearance). I have seen whole branches of the central artery such as the inferior temporal artery remain unchanged on the surface of a wide exudation that quickly led to scarring of the

retina. This was accompanied by chorio-retinitis, as witnessed by pigmentation which appeared later around each spot. The form of this sclerotic exudation is, as a rule, irregular. This distinguishes it from sclerotic chorio-retinitis, which forms small, round, more or less uniform spots all over the retina. In a few cases I have observed a very remarkable picture. The irregular area of exudation was surrounded at the periphery by comma-like new vessels. Were they newly-formed vessels or capillaries distended by the *vis a tergo*? It is difficult to decide. Most probably they are newly-formed vessels for one can follow their development almost day by day. In a patient whose fundus is represented in illustration No. 23 (see " Sclerotic retinal vessels ") it took me six weeks to decide whether I saw new-formed vessels or a haemorrhage. After seven weeks from its first appearance the exudation was smaller and a definite vascular ring was formed around it. Two weeks afterwards this ring started to contract and advance and eleven weeks after its appearance this exudation disappeared without leaving any trace.

This sclerotic exudation appears in advanced cases and may be quite independent of the height of even the diastolic blood pressure; it may be absent when the latter is at its maximum and appear when it has dropped a good deal. I did not see it in mild cases, the systolic blood pressure always being 200 or more, the diastolic 100 or more when the patients were *first* seen. The retinal vessels were always in an advanced state of sclerosis. This exudation may remain stable for weeks or even months and yet disappear without leaving a scar or any other trace in the majority of cases. In a minority, a whitish spot is left permanently and later this may become surrounded by pigmentation, a sign of a corresponding chorio-retinitis. It also forms whole fields of white atrophic areas sometimes closely resembling in their direction the course of retinal vessels, but one can never see any vessels. This exudation is, in my opinion, the result of insufficient oxygenation of the retinal cells. It does not mean an actual and permanent blockage of the capillaries and arterioles, for then it would not disappear and would lead to scarring. Most probably it means a combination of perivasculitis, endarteritis and actual constriction or spasm of the fine vessels—the same process that I discussed at length when describing the " silver-wire " artery. I say " most probably " because this is purely a microscopical problem and I am concerned here only with the ophthalmoscopic picture. It is possible that the retinal vessels are independent of the " selfishness " of the respiratory and vasomotor centres, which will constrict all other vessels of the body to a maximum to have their own free supply of oxygen and blood. Therefore, the spasm may be purely a local one—a reflex of a diseased vessel wall, and we have seen in the

paper on " Sclerotic retinal vessels " that this disease is always patchy, that even in the same circumference of a vessel a portion may be changed and the rest not affected. That is why I did not see spasm or contraction of a large adjoining vessel in these cases. It was not involved, as it certainly would have been if the contraction had been of a vasomotor origin. The condition corresponds to the localized vascular crises of Pal. These only occur in pathological vessels; they may even lead, according to Oertel, to actual gangrene of the intestine, if the mesenteric artery be affected. They are the cause of angina pectoris. They explain the numerous cerebral manifestations of sclerotic patients, which can pass away without leaving any trace or can lead to actual cerebral lesion—the exact parallel to what is seen in the sclerotic exudation in the retina. Be that as it may, it always means a diminished supply of oxygen, a *local* starvation of the retinal cells and a *local* limitation of their metabolism of vascular causation. It is very gradual, very slow in onset, although the last drop may suddenly cause an already full cup to overflow. *That is why the resulting acidosis is not sudden and not extreme, and the oedema of the retina is therefore so insignificant as not to be detected by the ophthalmoscope.*

In two cases I have observed this blocking of the arterioles affect a very large portion of the central part of the retina. In both of these oedema was absent or only slightly marked. One case I have fully described elsewhere. The other one (see illustration No. II) is that of a woman aged 72 years. She was seen in the out-patient department of the London Jewish Hospital on June 8, 1928, with translucency of her retinal vessels lost, copper-wire colour, and the light reflex dotted. Severe arterio-venous compression was present. There was a great deal of retinal exudation, but the vessels were not covered; they were clear. There were many haemorrhages of different forms. The small and round ones were situated near the macula. The *right* eye was more affected. Blood pressure 150/85. Urine contained no sugar or albumin. Blood sugar 0·1 per cent., blood urea 33 mg. per 100 c.c. The haemorrhages gradually disappeared in the right eye, and the exudation gave place to scarring of the retina, the round foci being surrounded by pigmentation. In the left eye, the same process was going on. Then, a week or so later, the patient presented herself with the left eye showing all the central part of the retina around the macula, an area three or four discs in diameter, whitish, not oedematous, being sharply demarcated from the rest of the retina, with a cherry-red macula in the centre. In the macula there was a large flat haemorrhage; small flat haemorrhages were scattered here and there in the whitish area. Central vision was lost. The same picture was seen a few weeks

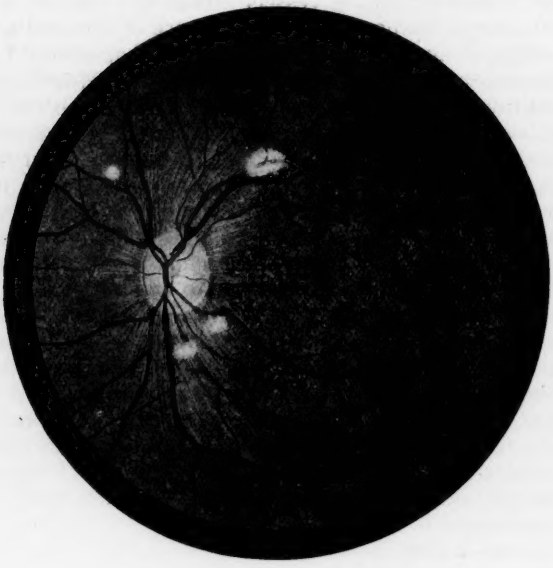


FIG. I.

afterwards. All this, in my opinion, was simply due to a blockage of the arterioles and capillaries. Why are the larger vessels clearly seen? Simply because they are situated superficially in the nerve fibre layer of the retina, whilst their branches pass backwards, deeper into the retina, to form there the end capillaries. The oedema of the tissue is too slight and too slowly progressive to go so far forwards and cover the large vessels also. The intermediate step between this kind of retinal exudation and real albuminuric retinitis I saw in a woman, aged 33 years, who had lost a tremendous amount of blood through a miscarriage. (Illustration No. I). About three weeks afterwards I examined her fundi. Translucency of the retinal vessels was lost, copper-wire colour, light reflex dotted, no white lines and no arterio-venous compression seen; a few slight retinal haemorrhages, many areas of exudation, irregular in shape, slightly oedematous, *i.e.*, not sharply demarcated from the surrounding retina. *This exudation covered some of the vessels exactly as in albuminuric retinitis. But the fact that there was no arterio-venous compression, or other signs of vascular disease, that the retina was healthy and clear and the vessels clearly seen and sharply defined immediately excluded the diagnosis of albuminuric retinitis.* Her blood pressure proved to be 140/75; the urine was normal. These two

cases together give a composite picture of albuminuric retinitis. In the first all the vascular components of albuminuric retinitis are present; in the second the element of oedema. This illustrates very well the complex aetiology of retinitis albuminurica—the vessel changes caused by hyperpiesis and oedema of the retina which may be independent of hypertension *per se*. Evidently in the above case the oxygenation and nutrition of the retina suffered

FIG. II.

suddenly and considerably with resulting oedema, because of the sudden loss of the oxygen carriers—the erythrocytes. A similar, but much more severe case of post-partum haemorrhage followed by retinal oedema was described by A. Fuchs (*Med. Klinik*, Sept. 28, 1928) with a striking and immediate improvement after transfusion of blood. This interference with nutrition also applies to retinitis albuminurica. In the latter the outstanding feature as already pointed out by Rochon-Duvigneaud is the oedema of the retina. *The " wool-patches " are of " fatty " colour; their margins are oedematous, gradually merging into the retina. If they are near to vessels they cover them. The vessels are not altogether clearly seen; they are not sharply defined but appear as if submerged in a tiny layer of water or covered with a very fine veil.* Sometimes only a few vessels look

like this; in severe cases nearly all of them. The disc may be very oedematous, but one cannot rely on this alone, as essential hyperpiesis may give a similar papillitis, because the sclerotic perivascular sheath may make the outflow of lymph very difficult. (The same mechanical explanation may suffice for many cases of papillitis or papilloedema of quite different origin and that is why the same picture of papillitis is common to many diseases and one cannot therefore rely solely on it in diagnosis. The retina, which does not stand in such a direct communication with the intracerebral pressure and whose lymphatic vessels are not so compressed, naturally does not suffer so much and therefore its oedema is not usually so complicated in its causation as oedema of the disc may be.) This oedema of the retina covering the vessels may vary from an inconstant veil that remains for a few days only and then disappears only to reappear again and again, or the oedema may be of a type that allows one to follow a medium vessel from the disc to the periphery and, in spite of all one's efforts, not be able to see some portion of it clearly. Usually the patient presents all the signs of advanced arterial sclerosis with slight exudation or haemorrhages in the retina. The blood pressure is very much raised; the urine has a specific gravity of 1,000 to 1,005 and contains traces of albumin or none at all. This oedema of the retina which covers the vessels is, in my opinion, the decisive factor in the diagnosis of retinitis albuminurica. If seen fully developed, it means that the prognosis is very grave and that most probably in a short time (at most, two or three years) the patient will succumb. Again I have to repeat that the ophthalmoscopic picture is only a part, although a very essential one, of the clinical whole. A considerably raised blood pressure is a *conditio sine qua non*. This oedema of the retina, very protean in character, presents so to speak a close analogy to all the finest gradations between the " normal " and the " inefficient " kidney of the vascular or sclerotic type and sometimes the ophthalmoscopic diagnosis may be extremely difficult.

Elsewhere I have suggested that nephro-sclerosis due to essential hyperpiesis may finally, when very advanced, be identical with malignant nephro-sclerosis or terminal stage of glomerulonephritis, *i.e.*, the stage of absolute kidney inefficiency. In the retina the above described peculiar phenomenon of oedema is the key to what is taking place. If the patient was originally a hyperpietic, this oedema will only indicate that the reserve power of the kidney is becoming exhausted. Altogether it is a very slow process, as every case of hyperpiesis is, and if by medication or dieting or in some other way the disease regresses, the patient may recover and the signs of " albuminuric " retinitis, especially of the " apoplectic " variety may disappear. Such a case is described

in my paper on " Arterial hypertension and retinal changes." In my opinion to such cases are applicable these words of Leber that " the retinal changes can regress if the basal pathological process in the kidney is healed, or is improved; in favourable cases even the highly advanced retinal changes can regress completely." (p. 817). Those are the cases that are described under interstitial nephritis as going on for many years—10 or 20 or not even suspected during life and suddenly discovered post-mortem. Hence the importance of the ophthalmoscopic diagnosis when it is made with all the foregoing reservations, for the presence especially of retinal oedema is a sure sign that the patient is fast approaching the stage of absolute kidney inefficiency.

I have watched a patient, aged 40 years, for two years, who at first shewed signs of vasculo-renal affection of the retina. The condition did not progress but on the contrary slowly improved.

June 1, 1927. Y. M., aged 40 years. Blood pressure 140/80. B. E. normal.

August 21, 1927. Severe back-ache. Blood pressure 210/110. Albuminuria, sp. gr. 1015. B. E. translucency lost, copper-wire colour, light reflex dotted.

September 9, 1927. Urine normal, sp. gr. 1007. Blood pressure 190/110. B. E. white lines on the crossings. Veins beginning to be crushed. Two small points of retinal exudation.

May 11, 1929. Blood pressure 200/120. Marked, but not advanced sclerosis of the retinal vessels. No oedema, haemorrhages, or exudation seen. " Feels fit."

In another case, that of a woman suffering from diabetes mellitus and described in my two previous papers, I was inclined to think that she was developing true interstitial nephritis, but she proved to have nephro-sclerosis. In spite of getting insulin and a normal blood-sugar, her blood pressure is 200—230/109. The blood urea in 1927 was 49 per cent. and urea concentration test 1·1 per cent. and 1·7 per cent. (first and second hours). Phenolphthalein test 6·6, 22·2, 4·4. Her retinae are full of haemorrhages and enormous scars. In 1928 her blood urea was 22 per cent., blood sugar 0·22 per cent. and urea concentration test 0·35 per cent. and 0·4 per cent. (first and second hours). She is in the same state of health now, 1930, but the destruction of the retina is enormous without actual and persistent oedema.

It is my impression that I have not come across a case of albuminuric retinitis without the blood pressure being considerably raised. That is why in my opinion only definite forms of nephritis, and not every type as Fuchs and Fox evidently think, are accompanied by retinitis albuminurica. Swanzy believes in the association of high blood pressure with retinitis; "The arterial tension is always high when retinitis is present, even in the young."

Parsons says (p. 351) that "Generally the history of severe headache may be elicited and the blood pressure is high, usually about 200 mm." The opinion of Foster Moore is that " It is very unusual to meet with retinitis except in the presence of a considerably increased blood pressure," and furthermore that " some degree of oedema of the retina is probably present in all cases." Rochon-Duvigneaud, as quoted by Bailliart, always found renal retinitis in association with considerable hypertension. And if one adds to the above authorities the partisans of the theory that retinitis albuminurica is entirely the result of vascular changes, as, for instance, Duke-Elder and Fox, then the overwhelming majority always find arterial hypertension present in combination with albuminuric retinitis. One must not forget, that even arterial hypertension is relative in its character: a reading of 160/95 is not very pathological of itself, but it may be so, if the normal one for the same person was 120/70.

It is a clinical fact that the urine most saturated with albumin is met with in " nephrosis," where the blood pressure is normal and retinal changes do not appear. On the other hand retinitis is seen in patients with nephritis associated with raised blood pressure and scanty or even absent proteinuria. Therefore the old term of " albuminuric " retinitis is not a satisfactory one: " renal " would be much more appropriate, although it implies that the aetiology of retinitis is dependent upon the kidney affection, a theory which, as will be shewn later, is not yet proved.

When the diagnosis of retinitis albuminurica is made, the changes are so advanced that it is impossible to state whether any healthy vessels are left. This and the fact that arterial hypertension accompanies the retinitis give support to the theory of vascular disease being the cause of retinitis. The first to maintain this view were Michel and Herzog Karl Theodor. On the other hand Rochon-Duvigneaud has shown that in many microscopic sections he could demonstrate retinal vessels sufficiently healthy to exclude the possibility of their being the cause of marked retinal changes. In this he is supported by Ginsberg, Schieck and many others. Of modern writers Fisher is, of course, entirely for the vascular theory, " The ophthalmologist who discovers ocular changes should not make the diagnosis of nephritis, but one of vascular disease, which may presumably be affecting also the patient's kidneys " (p. 631). " The retinitis is not a sequel of the kidney disease. In parenchymatous nephritis the retinitis is the result of toxaemia and therefore the " blindness " may disappear. In interstitial nephritis it is caused by insufficient oxygen supply to the tissue and is therefore mostly immovable." Duke-Elder holds (p. 191) " That the basis of pathology of all three (arterio-sclerotic, diabetic, and renal retinitis) is in all probability the same." Fox

writes " Although albuminuric retinitis may be seen in any form of kidney disease, it is encountered most frequently by the ophthalmic surgeon in connection with the cirrhotic kidney." (p. 313). He also refers to the veiling of the retinal vessels by the exudation. " Coincidentally with the changes in the retina there is a progressive degeneration of the vascular system, accompanied by high arterial pressure ...." Poulard is of Rochon-Duvigneaud's

FIG. III.

opinion that retinitis albuminurica is a disease *sui generis* and is not the result of vascular changes. Swanzy thinks that the retinal changes " are probably toxic in origin and not caused by arterio-sclerosis." Parsons writes: " There is no constant relationship between the retinitis and the vascular disease ..... The retinitis is, therefore, not directly due to the vascular disease, but probably to toxins circulating in the blood-stream."

Vascular disease of the retina, as characterized by accompanying hypertension, is of very slow development, although it commences very early. It presents a picture of gradually developing peri-vascular sclerosis and is really an adaptation of the body to the new conditions of the circulation. It follows the general rule that the parenchyma of an organ, where the vessels only are affected by sclerosis, may function pretty well for a long time, even for many

years. This applies to the hyperpietic retina as well as to the hyperpietic heart or kidney. It is surprising how far the retinal cells may accommodate themselves to extreme degrees of vascular anoxaemia without developing oedema, although they may be completely destroyed by a haemorrhage (See the illustration No. III, the late stage of thrombosis of the temporal superior vein). If the parenchyma of the retina is involved, the process is also a very slow one of gradual strangulation, gradual starvation, very patchy, as every sclerotic process is, and therefore is not accompanied by considerable oedema. In albuminuric retinitis the process is rapid and strong. It is a sudden invasion of the parenchyma as well as of the vessels. We shall therefore see in the vessels actual degeneration of the vessel wall more pronounced —" non-true "—silver-wire artery, haemorrhages and oedema of the wall; the perivascular sheath will be oedematous. In short, it will be a rough, deformed, aggravated vascular picture, instead of the fine, beautiful sclerotic vessels full of nuances. Thus the retina will be brick-red from hyperaemia, swollen, oedematous, with " wool-patches," macular star and large haemorrhages. It is a fact of minor importance and may be even a coincidence, as my number of cases is so small, but I never saw a true silver-wire artery; evidently, the vessels are attacked by a powerful toxin and in such a short period of time, that they are not capable of developing the more delicate signs of vascular degeneration. Only the more frequent use of the sphygmomanometer in the routine examination of patients and the regular, frequent and careful examination of the fundus in every case of hypertension can decide in our present state of knowledge the important pathological question of the cause of renal retinitis. It is my belief that the retina suffers *pari passu* along with the vessels, the same toxin attacking both of them, which makes a pre-existent renal lesion in the previous history unnecessary. Moreover, I shall later quote a case where the patient was suffering for a year from fully developed retinitis albuminurica and yet all his renal tests were normal. What reason then have we to incriminate the kidney as the cause of retinitis, if the kidney still functions normally? I am obliged to the courtesy of Dr. J. Burnford for seeing a young girl, aged 20 years, with arterial hypertension of advanced degree, albuminuria and nearly normal fundus. Therefore, I did not feel justified in making the diagnosis of chronic interstitial nephritis. Six months afterwards she came again with a fully developed and typical albuminuric retinitis.

In toxaemia of pregnancy we have another striking instance of the same state of affairs. There the retinitis is always preceded by hypertension. Both the retinitis and hypertension will rapidly disappear if the pregnancy is terminated. The kidney is

obviously as much a victim of the intoxication as the vascular system, and the pregnancy is undoubtedly the cause of all the mischief.

Why then of all the higher sense-organs is the retina the first and foremost to be involved? Three explanations are possible.

(*a*) The kidney has an endocrine secretion, which is in intimate association with the vascular system and with the brain, especially the retina. According to De Schweinitz, Zur-Nedden believes that toxin is elaborated by decomposition of the kidney substances, which toxin possesses a selective affinity for the retinal vessels. One wonders what Zur-Nedden means by " kidney substances "; if tubular cells, then nephrosis is free from retinal changes. At present, this attractive hypothesis is insufficiently confirmed to be accepted as an explanation.

Bailliart claims to have discovered a *local* vascular hypertension of the retina only out of all proportion to the increased brachial pressure in every case of retinitis albuminurica, even before the oedema and exudation appear. If confirmed, and Bailliart is a very good authority indeed, this fact would at least point to the retinal affection preceding that of any other organ of the body in its reaction to the kidney disease.

(*b*) If the reader will recall the complex structure of the retinal capillaries as demonstrated by Krückmann, he will realize under what difficulties the metabolism of the retinal cells is carried on. The blood has to pass through the endothelium and the basal membrane on which the former is situated to enter the perivascular channel. Here it mixes with the lymph, which flows in an exactly opposite direction from the retinal cells to the vessel. The slowed stream has now to pass through two or three strong glial membranes to reach the intercellular space of the retina proper. Most probably the *vis a tergo* exhausts itself completely in traversing all these obstacles and it is left only to the physico-chemical force of the colloids of the retinal cells to select and absorb the necessary substances out of the mixture of lymph and blood—a very fine and delicate process indeed. If the retinal cells are comparatively healthy and only the vessels are sclerotic, the metabolism may still be normal athough proceeding more slowly and with some difficulty. With increasing oxygen deficiency the retinal cells, half-starved, become oedematous, part of the poisonous products of their own metabolism—most probably acid in nature—being unable to find any outflow; with relief of any associated spasm of the vessels the crisis may be got over. *If, however, the metabolism of the cells themselves be impaired and altered* and, furthermore, the outflow of their waste products (more poisonous than usual) be also impaired by degeneration and continuous spasm of the vessels, the result will be that the

perivascular lymphatic space will become a real cesspool, and the diseased retina cells will lose to a considerable extent their fine selective and absorptive powers. *This means that in albuminuric retinitis is seen an actual perverted metabolism of the retinal cells, most probably going on in other parts of the brain and even in the body too, but apparently aggravated out of all proportion by the peculiar anatomical structure of the retinal vessels.* A good proof of the truth of this theory may be found in syphilitic affections of the retina. It is a well-known fact that syphilitic retinitis (*i.e.*, syphilis of the anterior layers of the retina) is very rare. On the other hand syphilis is a very common affection of the uvea. The difference cannot be caused by the blood supply, the arteria ophthalmica being the common main trunk. We must therefore look for the cause in the local condition of vascularization. Be the cause of syphilis the spirochaeta pallida or spore of this parasite which lives and develops in the endothelial cells, either must penetrate through the wall of the capillaries, fight its way against the stream of lymph and then pass through strong glial membranes. Is it any wonder then that such a large parasite as the spirochaete fails to get over all these obstacles? The same applies even more to the immobile bacillus of Koch, and again tuberculosis of the retina is very uncommon while choroiditis is not infrequent. In the uvea the vascularization is different and as a result the condition for infection is more favourable. The embryological similarity between the retina and the kidney is of some interest too. In both we can differentiate two parts, the proper parenchyma cells that are ectodermic in origin, and the mesodermic vascular and supporting tissue that grows in later. We have seen that in the retina both parts are segregated by a special membrane and that, as Weigert stated, the vascular system is treated by the central nervous system as a foreign body. Should similar histological conditions exist in the kidney, the Bowman capsule and its prolongation down the nephron corresponding to the glial retinal membranes, it would satisfactorily explain why the organ is so closely connected with the retina. So far as I know, nobody has succeeded in making the same fine and precise injection-preparations of the human kidney as Krückmann has done in the case of the retina. One has only again to point out the remarkable fact that syphilis of the kidney, like that of the retina, is extremely rare although the kidney is a " vascular " organ par excellence.

At a recent discussion on retinitis albuminurica, held at the 30th Congress of German Biologists and Doctors, all the speakers were unanimous that retinitis albuminurica is always accompanied by arterial hypertension, but opinions were sharply divided on the nature and aetiology of retinitis albuminurica, some claiming a purely vascular origin and others denying this. Volhardt main-

tained a view identical with mine, that retinitis albuminurica means a pathological local metabolism of the retinal cells. Like myself, he also believes that the chief mischief is caused by the disorder of local cellular respiration; but on many other points his views are different. So far as the retina is concerned the features of vascular sclerosis in essential hyperpiesis may be explained by fibrotic changes of the perivascular sheath and not by a maximal contraction. If this explanation be correct, then retinitis albuminurica means an actual pathological metabolism of the retinal cells in combination with a vascular disease.

(*c*) Nephritis is the actual *fons et origo* of retinitis albuminurica. This was the general opinion until quite recently and is widespread even now. However, the question may be viewed differently. It is quite possible that both the kidney and the retina may suffer from the same general toxin and the gravity of the prognosis simply depends upon the fact that all the other vital organs of the body—brain, heart, liver—are involved to nearly the same extent, but we cannot examine them clinically with the same degree of precision as we can do in the case of the retina.

Animal experiments have failed up till now to shew any definite connection between inefficiency of the kidneys and raised blood pressure and, consequently, retinitis albuminurica. On the other hand benign nephro-sclerosis *per se* does not give rise to retinitis albuminurica for many years and, if it does so, it is always questionable if some superadded infection be not the real cause. In nephrosis *per se* retinitis albuminurica does not appear either. Therefore, we have to presume that a combination of glomerulitis and nephrosis is the cause of retinitis. But though both glomeruli and tubular cells are always involved in every form of nephritis, retinitis does not always appear; this may only mean that not enough of glomeruli and tubules are involved, or that the involvement is not very severe. It may be argued that perhaps complete elimination of all of them by extirpation of the kidneys would cause retinitis and even to a greater degree hyperpiesis. However, this is not the case. Spence describes cases of renal dwarfism dying from uraemia, but with a normal blood pressure and the absence of retinitis. It may be that there *is a special pathological factor, or a combination of several pathological factors, which give rise to nephritis and retinitis alike.* And this is my opinion. In the majority of the text-books, including even McDonagh's, the kidney is the cause of hyperpiesis. I think it is nothing of the kind. I mentioned this elsewhere, quoting endarteritis obliterans as an instance. Briefly, my opinion is that hypertension is always caused by the muscular contraction of the vessels, and a sufficient number of them is always affected at the time when the kidney-vessels are involved too. The kidneys are as

innocent of arterial hypertension as the hypertrophied heart. On the other hand, it is my opinion that essential hyperpiesis is the cause of nephro-sclerosis, an opinion also held by Fishberg and Gray. Generally speaking, in glomerulo-nephritis we have an inflammatory and in nephrosclerosis a degenerative process of the nephron-vessels.

Clinically we can verify this theory by testing the kidneys as to their efficiency by the methods known to us to-day. These are: Volhardt's water test, McLean's urea concentration test, the estimation of urea and of creatinin in the blood, and the intramuscular injection of different dyes. All of them are a tremendous step forward, but the exaggerated hopes that they aroused have not been realized. In the great majority of cases retinitis albuminurica is, as shewn by these tests, combined with kidney inefficiency. The opinions of authorities are sharply divided as regards their prognostic value. If in retinitis kidney inefficiency were always demonstrated by the various tests, it would be difficult to deny that nephritis is the cause of retinitis, but if fully developed retinitis be present and the kidney efficiency tests are normal, then we may assume that the body is being poisoned by a toxin which is not a product of nitrogen metabolism. Every toxin is a complex protein so far as our present knowledge goes. It is possible that the retina is more sensitive and shews the pathological process earlier than the kidney, and that both are afflicted by a common foe. Such cases are not common, but I have had one under my care.

The patient was a man, aged 24 years, first seen in 1924. Urine: sp. gr., 1015; much albumin present. Blood pressure 180/120. In the fundi translucency of the vessels had disappeared; the arteries were copper-wire, and shewed dotted light reflex. White lines were seen at arterio-venous crossings, where the veins were crushed. The retinae were hyperaemic but not oedematous. A small haemorrhage was present in the left eye.

To this picture of retinal sclerosis there were added two months later exudates, wool-patches and oedema around the macula. At that time his kidney function was investigated at a hospital by means of intravenous indigo-carmine and was pronounced "normal." A month later there was a definitely established albuminuric retinitis. His blood pressure and urine were very much as before. A urea concentration test shewed 2·5 urea. In the meantime the patient was going steadily downhill, suffering from many attacks of minor uraemia, and a second investigation of his kidney function three months after the first still gave fairly normal results. (Blood urea 40 per mille; serum-indican negative; water concentration, excretion and dilution normal). The fundi shewed minor changes in the fully established albuminuric retinitis during this time, and four months later a third examination of kidney

function still gave a fairly good result. (Blood urea 38 per mille; urea concentration test 1·7 per cent., 1·8 per cent., 2·3 per cent., 2·6 per cent., before administration of urea, one hour, two hours and three hours after, respectively). A year after he was first seen he developed a right Bell's palsy. The blood urea at the time was only 26 per cent. and decapsulation of the right kidney was performed. The kidney was only slightly enlarged. In spite of some immediate improvement the patient died within six weeks. His blood urea rose from 77 per cent. to 188 per cent. within 17 days, and the patient died 17 days afterwards. Dr. J. Burnford kindly furnished me with the following report on the post-mortem examination of the kidney.

" This case was an example of a subacute nephritis, contracting. The kidneys were mottled, finely granular, fairly large. Microscopically—chronic interstitial inflammation (round cells in great numbers between the tubules) with some parenchymatous changes; some glomeruli degenerated and inflamed; some tubules cystic. Arteries thickened, especially in the tunica media."

Which is more important in such a case, for prognosis and treatment, the retinal picture or the kidney efficiency tests? Is it a case of gross cardio-vascular disease, benign nephro-sclerosis, or true interstitial nephritis (malignant nephrosclerosis)? McLean is a staunch advocate of the kidney efficiency tests, even though in the experience of others they are not altogether reliable. " Indeed, cases of alleged chronic interstitial nephritis have been described, in which renal tests gave good results quite incompatible with the supposed conditions of the kidneys, and those anomalous results have been brought forward as an indication that the renal tests are not always reliable. In such a case it is the diagnosis that is at fault and not the tests." (p. 78). Elwyn holds that retinitis albuminurica, especially if the " star " around the macula be present, is a sure sign that the patient is fast approaching the stage of absolute kidney inefficiency, although the blood chemistry be normal. But though it is true that retinitis bears no direct relationship to the blood chemistry, one is not warranted in holding as Parsons does that " The degree of retinitis bears no fixed relationship to the nature or intensity of the renal mischief," a view shared by Fuchs: " The severity of retinitis bears no fixed proportion to the intensity of kidney disease .... the like is true of the subsequent course; the retinitis may improve while the kidney lesion grows worse or *vice versa*."

(To be Continued.)

THE INTRA-OCULAR PRESSURE AND DRAINAGE OF THE AQUEOUS HUMOUR

BY

FREDERICK RIDLEY

LONDON

(Received for publication December 11, 1930)

IN a recent article in this Journal, Duke-Elder has discussed my work upon this problem. As many of those addressed may not have read my paper, which was published in *The British Journal of Experimental Pathology*, 1930, attention may be drawn to some points not clearly set out by him.

In the second paragraph he refers to my introduction in which existing theories of aqueous drainage are discussed. I am aware that "the failure of particulate matter to pass through the endothelial cells of Schlemm's canal has no relation to the passage of fluid." This observation has no bearing upon the context of my paper. Speaking of Thomson's speculation, "that the lymph spaces around the angle may be provided with a system of valve-like openings," I quote the work of Nuel and Benoit (1895) who showed that the minute particles of indian ink do not pass into the canal of Schlemm from the anterior chamber, and conclude that such valve-like openings cannot exist. It is held that I have "failed to show that particulate matter is delivered out of the eye through the cornea." I did not attempt such a task.

Throughout the article I am rebuked for failing to recognise previous work. Where a previous worker has employed a similar technique and in all cases of quotation or reference, the reference is given. I have not given detailed references to the large number of workers upon familiar subjects.

My paper brings forward experimental evidence that the normal living cornea is permeable to diffusible substances and that the osmotic pressure due to the tear protein and the hydrostatic force exerted by the intra-ocular pressure determine the constant passage of fluid through the cornea.

Against these experiments, Duke-Elder "puts forward one only which is to his mind conclusive." An experiment in which methylene blue is introduced into the anterior chamber of a dog at a pressure not more than 2 mm. Hg above the normal intra-ocular pressure is described. After one and a half minutes the blue appears in the episcleral veins. This experiment is held to prove conclusively that the cornea is not an available channel of exit since no blue is seen in the cornea or conjunctival sac in one and a half

minutes. It is certain that the blue will not pass through the cornea in this time. No such claim has been made. Indeed a radicle so small as ammonium does not pass through in measurable amount in less than 15 minutes. As a refutation of my work, this experiment is irrelevant.

For a critical examination of experiments of this kind, reference may be made to Duke-Elder, 1927, *a*. where the conclusion is reached that such experiments may "prove anything or nothing," and to Duke-Elder, 1929, *a*. The experiment is nevertheless held to prove that the blue has passed into the episcleral veins *via* Schlemm's canal. It does not do so, all it proves is that if the experiment is performed blue will appear in the episcleral veins in one-and-a-half minutes.

Duke-Elder has shown by direct measurement in the dog that "The pressure in the exit veins is normally a little higher than the chamber pressure" and that "On raising the chamber pressure to a considerable height the pressure in the veins was found to rise coincidently" (Duke-Elder, 1929, *b*). Not until a chamber pressure of 40 mm.Hg is reached does the exit vein pressure fall to 39 mm.Hg by comparison. Since the chamber pressure in this experiment is raised not more than 2 mm.Hg the exit vein pressure is not less than the intra-ocular pressure. We must conclude that under the experimental conditions laid down fluid cannot pass into the episcleral veins via Schlemm's canal.

But it is stated that the blue appears in the episcleral veins in one-and-a-half minutes. The explanation is probably to be found in the fact that, "In the lower mammals, *e.g.*, the dog, the venous anastomoses are large and most of the blood from the eye is returned to the extracranial venous system, while in man it normally flows into the intracranial system" (Parsons, 1904). In the dog there is a free anastomosis between the circle of Hovius and the vortex veins themselves (Duke-Elder, 1929, *c*.) The venous arrangements in the eye of the dog are such that blue will appear in the episcleral veins whatever may be its site of entry into the venous system. "As far as they have been investigated (methylene blue has been discussed) all substances seem to enter and leave the eye by a process of simple diffusion" (Duke-Elder, 1929, *d*.) The blue which appears in the episcleral veins might have entered the vascular system in the eye of the dog by diffusion into *(a)* The canal of Schlemm; *(b)* The whole area of the capillaries of the ciliary body and iris. In the latter case the dye, having passed by diffusion into the capillaries, will be carried by the blood stream to the site at which it is observed. In the case of Schlemm's canal not only is the diffusion area minute by comparison but we have seen that there is no fluid stream to carry the blue. There can be little doubt that

in this experiment the blue has passed into the episcleral veins *via* the capillaries of the ciliary body and iris. The experiment is not a refutation of my work since it has no bearing upon it, and what Duke-Elder, in his article, describes as obvious is at variance with his own experimental work and teaching and, to say the least, highly improbable.

I do not deny that a swelling of the vitreous may occur under experimental conditions of abnormal pH or salt concentration. My paper states that "there is no evidence that the variations in these factors necessary to cause such a volume change in the vitreous (of a living intact animal) occur." Duke-Elder supports this strongly. "The pH of the blood is a factor of extreme constancy." "Any treatment which will change the reaction of the blood to any marked extent upsets so extensively the essential functions of the experimental animals employed that observations of the intra-ocular pressure in these circumstances are of little value." (*Trans. Ophthal. Soc. U.K.*, 1929, p. 102.)

Also Adams and Kerridge (1930) have shown that the pH of the vitreous in rabbits fed on naphthalene, in which extreme vitreous degeneration may occur, is remarkably constant. Similarly, I do not know of any evidence that the salt content of the blood in man falls, either in health or disease, sufficiently to cause a significant swelling of the vitreous at normal pH. In "The nature of the vitreous body" (Duke-Elder, 1930), we read on page 49 that if the vitreous swells it does so by absorption of aqueous humour. It is important to observe that swelling of the vitreous can never produce an increase of volume of globe contents so long as any aqueous remains. If the vitreous swells the volume of aqueous plus vitreous decreases (Lewis, W.C., 1919) so that the volume of globe contents would be diminished by swelling of the vitreous and the intra-ocular pressure would fall; shrinking of the vitreous, on the other hand, might give rise to an increased intra-ocular pressure if the rate of shrinking were so rapid that fluid could not pass out of the eye into the capillaries sufficiently rapidly to compensate the small increase of volume of aqueous plus vitreous. In his article, and in many instances in his writing, my critic suggests that the passage of diffusible substances through a permeable membrane need bear no relation to the passage of fluid. It does bear this constant relation, that, where diffusible substances in water solution can pass through a membrane, water must do so if the hydrostatic and osmotic forces on either side of the membrane are not balanced. My paper states in a series of six equations the equilibria, both osmotic and hydrostatic, that must exist in the eye if fluid is not to enter or leave the capillaries of the uvea, for the intra-ocular pressure existing at the time of observation. The fact that membranes permeable to

diffusible substances in solution in water are permeable to water itself is essential to an understanding of the problem under consideration.

The observation that hypotension may occur with maximal vaso-dilatation is familar in the experiment of opening the anterior chamber. Duke-Elder's reference is vague (Duke-Elder, 1926-29). In the experiment in which 30 per cent. saline solution is injected into an experimental animal the eye is subjected to an active dehydration and it is reasonable to suppose that vaso-dilatation is unable to counteract this decrease of volume of globe contents, as in the simpler experiment described above.

Duke-Elder says "section of an eye with chronically raised tension frequently shows a thin and atrophied choroid, ciliary body and iris, the whole uveal tract being obviously pressed almost out of existence by something else—the vitreous." Fuchs attributes this condition to "the atrophy which, after the subsidence of the early congestive symptoms, sets in in all parts of the uvea," and proceeds to describe the pathological changes, including fibrosis and vascular changes, which lead up to this end result. These changes cannot be described as a simple pressure atrophy.

The last paragraph in the criticism confuses the issue. Many workers have advanced evidence as to the composition, osmotic pressure, conductivity and other properties of the aqueous humour as compared with the blood plasma which suggests strongly that the aqueous is formed by a process of dialysation from the blood. My paper draws attention to the fact that such a procsss may be either "static dialysation," if the aqueous is stagnant, or "pressure dialysation" if the aqueous is subjected to a constant drainage, *e.g.*, through the cornea. When we come to consider the possibility of applying Donnan's theory of membrane equilibria to this problem we have to remember, *inter alia*, the following reservations—

1. That Donnan's simple formula is not applicable to a system in which a protein containing fluid such as plasma, is separated from a protein free fluid such as aqueous, without suitable correction.

2. That it cannot be applied unless the assumption is made that the aqueous is stagnant.

Duke-Elder has attacked this problem by:—1. A full analysis of horse aqueous and serum. From these figures he claims to have demonstrated the following

a. That there is an unequal partitioning of the anions and cations between the aqueous and the blood plasma.

b. That this unequal partitioning is in accordance with Donnan's theory.

c. That when the percentages of sodium and chlorine in particular are converted into concentration terms and the figures substituted in Donnan's simple formula :—(Duke-Elder, 1927, *b.*

$$\text{viz. } [Na]_{aq.} \times [Cl.]_{aq.} = [Na]_{blood} \times [Cl.]_{blood}$$

$$\text{„ } 121 \times 123 = 145 \times 103$$

$$148{\cdot}83 = 149{\cdot}35$$

the apparent discrepancy of 0·3 per cent. can reasonably be explained—"as being within biological limits—"*It is* unjustifiable to hope for results of any more approximate accuracy." Considering these results in conjunction with the fact that the relationship holds good only for fluids which are stagnant, he concludes that "the aqueous is *without an active circulation* in the generally accepted sense of the term but rather it is essentially stagnant" (Duke-Elder, 1927, *c*).

2. Subsequent micro-analyses of sugar and chloride in aqueous and arterial and venous blood taken from the same animal at the same time are put forward in support of the figures and conclusions obtained from the major analysis. These micro-analyses only strengthen the main argument, however. They are incapable, alone, of supporting the belief that a Donnan equilibrium exists, for they do not supply information with regard to a pair of anions and cations such as sodium and chlorine. He returns to the figures derived from his major analysis when discussing the significance of those obtained in his micro-analyses. (Duke-Elder, 1927 *d*.)

My paper claims to prove that there is a constant drainage of fluid out of the anterior chamber through the cornea in the normal living eye. If this is so it is clear that the aqueous is not stagnant. Duke-Elder's work and mine are thus opposed.

Notwithstanding his complaint that this major analysis is "only a rough estimation" it remains the only evidence he has advanced which properly supports his claim to have demonstrated a Donnan equilibrium between the aqueous and the blood. This, in conjunction with the fact that the percentages of all the more important constituents are given to the fourth decimal place and that a paragraph is devoted to explain a discrepancy of 0.3 per cent. as being within the limits of biological error, appears to justify the careful consideration given in my paper to this analysis and the conclusions based upon it. This major analysis is open to the following criticism :

1. It involves the comparison of a pooled sample of horse aqueous with a single sample (not similarly pooled) of horse serum. The composition of blood varies within wide limits; thus for man the normal chlorine content varies from 340 to 380 mgs. per 100 c.c. (blood plasma), sodium 325 to 350 mgs. per 100 c.c., sugar 60 to 180 mgs. per 100 c.c. It is reasonable to suppose that variations of a similar order of magnitude occur in the horse. The figures given by a single blood analysis may therefore involve quite large errors.

2. The comparison is made between aqueous and serum but the fluids it is attempted to compare are aqueous and capillary blood plasma. The following discrepancies are inherent.

(a) Capillary blood approximates to arterial rather than to venous blood. In passing from the arterial to the venous state a chloride shift occurs so that the chlorine content of venous plasma expressed as NaCl may be 342 mgs. per 100 c.c. as compared with 349 for arterial plasma; the serum is derived from venous blood and an error occurs in the chlorine estimation for this reason. It is to be noted that the sodium content of the plasma is not affected by the chloride shift. A greater discrepancy arises from the fact that "when blood is shed a chloride shift occurs from corpuscles to plasma, *in vitro*, unless escape of blood gases is prevented." (Harrison, 1930. Chemical Methods in Clinical Medicine, p. 173. The foregoing figures are derived from the blood analysis given on p. 305). In addition to these objections there is the fact that if a Donnan equilibrium exists between the aqueous and the blood then, substituting concentration terms in Donnan's simple formula (***supra***) the product [Na] × [Cl.] for the blood should exceed that for the aqueous by about 4 per cent. They should not be equal as Duke-Elder found them to be. For these reasons the writer does not accept the conclusion that there is normally no drainage of the aqueous.

With regard to the estimation of the osmotic pressure difference between blood and aqueous in which he obtained figures agreeing closely with those given by other workers using various methods I have preferred the work of Dieter (1925) because his investigation was well controlled and he used the depression of freezing-point technique. In this experiment the control is open to the criticism that the estimations of the conductivity and glucose-content of the aqueous show significant variations in the course of the experiment in many instances. The refractive index of the aqueous before and after the experiment was found to be the same to the sixth decimal place in six instances, but it is to be remembered that the Dipping refractometer employed has an instrumental error of $\pm$ 3.7 at the fifth decimal place. (Duke-Elder, 1927 *e*. and 1930 *a*.) With regard

to the method many workers consider that the depression of freezing-point technique is preferable.

Duke-Elder states that my criticism of the application of the simple formulae of Donnan challenges an enormous amount of work done by physiologists and biochemists for the last two decades. I have referred this matter to Mr. G. S. Adair who states that "in the form given in my paper the criticism of Donnan's equation is in agreement with the most recent work on membrane equilibria, which shows that there are considerable deviations from the simple formulae of Donnan. Reference may be made to the papers by Van Slyke, Hastings, Murray and Sendroy (*Jl. Biol. Chem.*, Vol. CXX, p. 701, 1925), and by Adair (*Proc. Roy. Soc. A.*, Vol. CXX, p. 572, 1928), which show deviations of the order of magnitude referred to by Ridley."

REFERENCES

Adams and **Kerridge.**—*Brit. Jl. of Ophthal.*, Vol. XIV, p. 397. 1930.
Dieter, W.—*Arch. f. Augenheilk.*, pp. 96, 179. 1925.
Duke-Elder, W. S—The Nature of the Intra-ocular Fluids, pp. *a*. 81, 82, 83 : *b*. 69, 70; *c*. 77; *d*. 31; *e*. 34; *f*. 72; *g*. 54. 1927.
——— Recent Advances in Ophthalmology, pp. *a*. 176, 177; *b*. 181; *c*. 179; *d*. 201. 1929.
——— The Nature of the Vitreous Body, *a*, p. 40. 1930.
Fuchs.—Text Book of Ophthalmology. Eighth edition.
Lewis, W. C.—A System of Physical Chemistry, p. 356. 1919.
Nuel and **Benoit.**—*Arch. d'Ophtal.*, p. 41. 1895.
Parsons, J. H.—The Pathology of the Eye. London. 1904.

ABSTRACTS

I.—REFRACTION AND OPTICS

(1) **Ferree, C. E. and Rand, Gertrude (Baltimore).—Intensity of illumination and other factors influencing the sensitivity of the radial test for astigmatism.** *Amer. Jl. of Ophthal.*, October, 1929.

(1) **Ferree** and **Rand** find that the sensitivity of radial charts for detecting astigmatism can be much increased by raising the intensity of their illumination. The illumination should, if possible, be limited to the area of the chart, the surroundings being in subdued light. This can be done in one of two ways—the chart may be an opaque one, illuminated in front by a projecting lantern

which throws a disc of light on to it, or the chart may be painted on lightly ground glass and illuminated from behind. It is advised that two charts be used, one bearing radial lines, and the other a simple cross, which can be rotated into any desired position, one of the limbs being surmounted by a Maddox V. If the chart is to be used at 20 feet, the width of the lines should be 7 mm., the length of the radii being about 12 cm. By varying the illumination two sets of curves were obtained. The first of these gives the relation between the illumination of the chart and the amount of astigmatism the patient could detect. An error of 0·37D. could be detected with the minimum illumination, 0·25D. required about 3 foot candles and 0·12D., 25 foot candles. The second curve gives the amount of illumination required to detect given amounts of error in placement of a 0·25D. cylinder when correcting an astigmatism of this amount. At 5 foot candles the error was as big 40°, whereas at 50 foot candles it was only 5°. The ideal to be aimed at is, therefore, to have the intensity of illumination of the chart about 50 foot candles while the illumination of the surrounding objects is not more than 3 foot candles.

F. A. W-N.

(2) **Cattaneo, D. (Rome).—Observations on the mechanism of accommodation. (Quelques observations sur le mécanisme de l'accommodation.)** *Ann. d'Ocul.*, CLXV, p. 32, 1929.

(2) In repeated examinations, with the slit-lamp microscope, of coloboma of the iris, **Cattaneo** was able to satisfy himself

(1) That the fibres of the zonule were attached for the greater part behind the ciliary processes, a few in the hollows between the processes and none to the processes themselves;

(2) That the ciliary processes moved mesially towards the equator of the lens during accommodation;

(3) That the fibres of the zonule were definitely relaxed during accommodation, whether this was produced voluntarily or by the action of eserine;

(4) That the lens was subject to trembling movements, no doubt the result of the zonular relaxation;

(5) That the space between the equator of the lens and the projection of the line of limbus was increased, and that the lens was displaced to the right or to the left when the head was tilted to right or left, and that this movement was greater below (pendulum-like);

(6) That the transverse or equatorial diameter of the lens was lessened at the same time.

He also noted that the effects of eserine instillation were rapidly abolished by one instillation of atropine and that as many as twelve instillations of 1 per cent. eserine after that failed to cause a repetition of the accommodative effects of eserine during repeated examination for two hours.

HUMPHREY NEAME.

(3) **Jackson and O'Rourke (Denver).—Practical accuracy and convenience in trial lenses.** *Jl. of Amer. Med. Assoc.*, January 11, 1930.

(3) In this paper **Jackson** and **O'Rourke** deprecate the blind purchase by the young ophthalmologist of a trial case containing lenses which have been selected by a manufacturing optician. The optician usually supplies spherical lenses which are biconvex and biconcave, whereas they should certainly be plano-convex and plano-concave. Some opticians in London will supply a full set of these latter without any extra charge, others charge about 20 per cent. more. All cylindrical lenses are, of course, plano-convex and plano-concave.

The kind of practice which is to be undertaken should also influence the number of lenses which will be required, for if the trial case is not to be in constant use the 300 lenses of a complete case may be reduced by nearly one half. The selection of a suitable trial frame is of the greatest importance, and the authors rightly emphasise the desirability of lightness, and themselves use steel frames weighing half an ounce only, which are without a scale for reading the axis of the cylinder, this being done with a protractor. However, the reviewer prefers a frame which is now on the market weighing less than two ounces, having slots for the reception of three lenses, each section of the frame being adjustable as regards distance from the nose.

A. F. MACCALLAN.

(4) **Nicolato, A.—The causation of myopia. (Della genesi della miopia).** *Arch. di Ottal.*, November, 1929.

(4) **Nicolato** reviews all the suggested causes of myopia and finds them all insufficient; he concludes that there must be some underlying condition to which the suggested causes are, at most, adjuvant. He suggests that this state has to do with the action of an endocrine gland and picks out the thymus as that most likely to be the true cause; he argues that the thymus is most active at the time of life in which myopia commonly develops; further that experiments on animals show that after the removal of the thymus, there are changes in metabolism, especially with regard to calcium salts, and also delay in physical and psychical development, which he thinks paralleled by the condition of many young myopes.

The records of myope classes ought to give much help towards solving the question of the influence of near work on myopia; if it is true, as most surgeons believe, that the use of the eyes for near work is dangerous in myopia, the children who attend myope schools and classes ought to show an average increase less than that of similar children who have not the advantages offered by these special classes; the publication of statistics on this point is much to be desired.)

HAROLD GRIMSDALE.

(5) **MacCallan, A. F. (London).—Report on a series of 100 consecutive refraction cases with full vision after correction. Indications for reference to the branches of the medical profession of those showing evidence in the eye of toxic conditions.** *Trans. Ophthal. Soc. of the U.K.*, Vol. XLVIII, p. 181, 1928.

(5) **MacCallan** has attempted to show the incidence of infection from a primary focus (such as the teeth, tonsils, etc.) in 100 consecutive cases suffering from errors of refraction. This series of cases are private patients and in all of them the vision after correction was 6/6 or 6/5. 58 per cent. were under 40 years of age, and besides a small error of refraction some of them suffered from other ocular changes such as blepharitis, conjunctivitis, episcleritis, lenticular and vitreous opacities, retinal haemorrhages, hyperaemia of the retina, effusion and degeneration at the macula, and dacryocystitis.

In all these cases a primary focus of infection was sought for in the hope of dealing with it and thereby checking and curing the ocular changes and improving the general health.

Forty-two patients were referred to a dental surgeon; 33 of these were radiographed and 30 showed pathological changes in the teeth and jaws. 22 had apical abscesses, in one there was a dental cyst with an apical abscess. The remaining 19 patients suffered from dental conditions requiring surgical intervention.

One patient had the appendix removed for chronic appendicitis; 2 underwent tonsillectomy for enlarged and septic tonsils; and 1 had a suppurating antrum of Highmore with septic teeth.

Thirteen patients received medical and dental treatment, and 7 refused medical and dental advice. The examination for a primary focus of inflammation was incomplete in 8 cases.

Twenty-six patients had ametropia unassociated with any discoverable focus of infection.

H. B. STALLARD.

II.—PERIMETRY

(1) **Ferree, C. E. and Rand, G. (Baltimore).— Methods for increasing the diagnostic sensitivity of perimetry and scotometry with the form field stimulus.** *Amer. Jl. of Ophthal.*, February, 1929.

(1) **Ferree** and **Rand** have been working for many years on perimetry, and in this paper discuss methods for improving the sensitiveness of the instrument. One is to use coloured objects instead of white ones since the size and shape of the colour fields are more affected than those of the form fields by factors influencing the sensibility or response of the retina. There are, however, many objections to the use of coloured objects for perimetry, which render them impracticable for clinical purposes. Another method is to use very small objects, but here the influence of the acuity factor particularly in the peripheral portions of the retina may operate in such a way as to be detrimental to the purpose of the test. Reduction of illumination may be employed, but this demands complex apparatus, and a more simple procedure is to reduce the contrast between the test object and the background. This can easily be done by having objects painted in various shades of grey and using them on a black screen. A size of 0·5° is suggested as suitable for the object. Care has to be exercised to distinguish the small depressions of sensitivity which occur in a normal field from very faint scotomata, but fortunately the areas of the retina in which scotomata are most frequent are not those in which these normal depressions of sensitivity are likely to occur.

F. A. W-N.

(2) **Gaudissart, P.—I. Quantitative perimetry. II. Perimetry with coloured objects. (I. La périmétrie quantitative. II. La périmétrie avec index colorés.)** *Ann. d'Ocul.*, Vol. CLXV, p. 177, 1929.

(2) **Gaudissart** states that it has been generally accepted that the use of coloured objects in perimetry gives greater precision than the use of white. This has come about owing to the fact that a coloured object of small size has been much nearer to the limit of perception than a white object of the same size. The visual field for a 10 mm. red object at 30 cms. distance extends normally out to the 50° or 60° circle. For a white object the same result, in similar conditions but with a 2·5 mm. white object, is obtained at 100 cms. distance. The great difficulty, however, even with an intelligent patient, under examination with a coloured object is to decide when the colour is perceived. A red object produces first an impression

of light, and then passes through yellow, orange and light red to vivid red. Gaudissart refers to the work of Ferree and Rand on the relationship of illumination intensity and the size of object upon the field. With fifty-one "foot candles," the field of red is up to 85° in the temporal horizontal meridian and 55° in the nasal. With seven "foot candles" the dimensions are 40° and 30° respectively. Gaudissart emphasises the importance of the quality of the colour. Variations occur in degree of saturation and in purity of colour. He employs a cubical box with a circular 10 mm. aperture in the side. It contains a 16 candle power lamp, filtered by a screen placed against the hole (Lifa Lichtfilterfabrik-Ausbourg Nos. 900 and 901 give a pure red of average 6,600 Angstrom units). Low intensity of light on the background tends to increase the size of the colour field. By this apparatus it is found that red 10/1000=white 2·5/1000 (*i.e.*, that similar results in the normal are obtained by a 10 mm. red object as with a 2·5 mm. white). Charts are published illustrating the similarity in the fields for red and white objects when proportionate sizes are used as indicated above, in the study of cases of pressure by a chiasmal tumour, of tobacco amblyopia and of retrobulbar neuritis.

(The abstract of a previous paper by the same author was published in the *Brit. Jl. of Ophthal.*, Vol. XII, p. 341, 1928).

HUMPHREY NEAME.

III.—OPTIC NERVE AND OPTIC TRACTS

(1) **Weill, Georges (Strasbourg).—The relationship between inflammation of the posterior sinuses and disease of the nervus opticus.** *Arch. of Ophthal.*, March, 1929.

(1) After mentioning various conditions such as orbital phlegmon, which may originate from sinus infection, **Weill** proceeds to define the terms of his discussion, and limits himself to acute retrobulbar neuritis. This disease which occurs amongst young people, principally females, is generally unilateral and is marked by sudden and serious diminution of vision with a central scotoma and no obvious changes in the fundus "a disease in which neither the patient nor the physician sees anything." As a result of a prolonged study of the literature, the author finds (1) that the rhinologist practically never observes acute retrobulbar neuritis associated with posterior sinusitis (2) that the ophthalmologist practically never sees lesions of the sinuses in cases of acute retrobulbar neuritis. On the other hand, surgical intervention has often produced rapid and definite improvement of vision, but the author is not inclined to

attach much significance to this since out of 40 of his cases 85 per cent. have obtained almost complete restoration of vision without surgical intervention. The most likely explanation of the typical cases is that the condition is an early manifestation of multiple sclerosis. The other signs of this condition may not be in evidence, but in a fair number of these cases, the abdominal reflexes are absent. Cases of atypical retrobulbar neuritis on the other hand often emanate from a lesion of the sella turcica or of the sphenoidal region. Operative interference in cases of typical acute retrobulbar neuritis is therefore to be deprecated.

F. A. W-N.

(2) **Hogg, G. H. (Tasmania).—Whooping cough and optic neuritis.** *The Medical Journal of Australia*, August 3, 1929.

(2) **Hogg** reports a case of secondary optic atrophy seen in a child, aged 3 years, and following upon an attack of whooping cough, during which attack the child had complained of visual symptoms and shown definite signs of ocular discomfort. One eye was blind, but power to see objects, such as large toys, was preserved in the other. The only other recorded cases, seven in number, are listed. All but one of these were seen in the stage of optic neuritis, which resulted in complete recovery in several instances.

R. C. DAVENPORT.

IV.—MISCELLANEOUS

(1) **Edridge Green, F. W. (London).—Subjective phenomena and colour vision.** *Lancet*, September 7, 1929.

(1) In this letter to the editor of the *Lancet*, **Edridge-Green** states that it summarises a communication read last July before the Association Française pour l'Avancement des Sciences at the Congress in Havre. The letter is therefore really in the nature of a scientific note. The author commences with a criticism of the physicists "who treat the old theories of colour vision as if they were demonstrations in mathematics," and, more particularly, finds fault—and rightly—with the words of a well known physicist (not named), "When we are dealing with something definite and tangible such as colour," etc. As the author points out colour is not definite and tangible. He then proceeds to the description of after-images which may be seen best on awaking in the morning after looking at the window or windows. It is impossible to abstract this interesting description which deals with the appearance of currents

circulating in the centre of the retina, which currents are not due to the retinal circulation. Edridge-Green next comes to what is probably the main point namely, that these observations support his theory of vision (1889) that the cones are the sole terminal perceptive elements, the rods being concerned with the formation and distribution of the visual purple. He asserts that the duplicity theory (rods as the percipient elements in a dim light) is supported almost entirely by mis-statements namely, (1) that certain animals have only cones and others have only rods; (2) that the periphery of the retina is colour-blind; (3) that the eye is totally colour-blind in dark adaptation; (4) that the Purkinje phenomenon and the recurrent image are not found at the fovea. The author denies every one of these statements and gives his reasons for such denial: (1) Even the most quoted example, the tortoise, has clearly defined rods and cones; (2) the periphery of the retina is not colour-blind when colours of sufficient intensity are used; (3) in dark adaptation the eye is not totally colour-blind. Further, there is no scotoma or blind area corresponding to the rod-free portion of the macula which is equal to a visual angle of about three degrees; (4) the Purkinje phenomena and recurrent image are found at the fovea. Hess showed not only that the recurrent image was found at the fovea, but also that it was bent outwards at this region as it would be if the visual purple had to flow into the fovea.

ERNEST THOMSON.

(2) **Morax (Paris).—Stereoscopic colour photography of the anterior part of the eye. (La photographie stéréoscopique en couleurs du segment antérieur).** *Ann. d'Ocul.*, Vol. CLXVI, p. 261, 1929.

(2) **Morax** states that interesting records of various conditions have been made during recent years by stereoscopic colour photography. The Zeiss Drüner camera is used, with autochrome plates 6·5 × 6 cms. A special magnesium powder lamp is used which is caused to flash by means of a short circuit through a fuse. The patient's head is supported on a chin support and forehead rest as for the corneal microscope. The F. 55 pair of lenses of the Zeiss eye microscope is employed. A special colour filter (by M. Montpillard) is required with the magnesium light (from "Poudre Gaulois"). The lamp with a thick glass protector is placed close to the patient's face. Focusing is performed with illumination from a filament lamp placed in such a position that corneal reflections do not obscure the object of the photograph. The magnesium lamp is placed in the same position and fired. Three-colour reproductions of the lumière stereophotographs are published.

HUMPHREY NEAME.

(3) **Evans, J. N. (Brooklyn).—A scotoma associated with strabismus.** *Amer. Jl. of Ophthal.*, March, 1929.

(3) **Evans'** paper concerns the findings in 16 cases of strabismus. Most of these were "cosmetically straight" but had an amblyopic eye of visual acuity varying in the different cases from 6/60 to 6/15. The apparatus employed was the Lloyd stereo-campimeter which allows of binocular fixation. A green filter was placed before the fixing eye and a red one of equal luminosity before the amblyopic eye, the objects used being small silver wires fused at the end into 1 mm. balls and whitened with hydrochloric acid, the stem and handle of course being black. Using this method, in each case, an absolute central scotoma was found two degrees in diameter, whose centre occupied the fixation point and whose temporal border was connected with the blind spot by a narrow horizontal angio-scotoma. The author then attempts to correlate the presence of this scotoma with the defective visual acuity. He found that a test object consisting of a black strip tipped on one side by two white squares of one minute size separated by an interval of one minute was unrecognisable as such outside the one degree circle. He deduces therefore, that a scotoma extending one degree in each direction about the fixation point, or two degrees in diameter, would render unrecognisable a letter whose limbs measure two minutes in width; such a letter is the 6/12 one. The average visual acuity of the amblyopic eye in the patients examined was, however, 6/22, a fact which is accounted for (1) by the wandering movements of the amblyopic eye in the endeavour to read the test type; (2) that to perceive the whole of the letter its centre would have to fall on the retina outside the 1° circle and so on a less sensitive portion of the membrane. An interesting explanation is given of the statement often made by patients, when their amblyopic eye is being tested, that the letter flashes in and out again as they are examining it. Such a patient learns to fix with a part of the retina just peripheral to the border of the one degree defect. This part of the retina soon becomes exhausted and the patient moves his eye so that the image shall fall on a point diametrically opposite, and in doing so, the image passes over the scotomatous area and so is lost for a short time. The presence of the scotoma is attributed by the author to defective uncovering of the macula, during development.

F. A. W-N.

(4) **Alvis, B. Y. (St. Louis).—Amaurosis following ingestion of ethylhydrocuprein.** *Arch. of Ophthal.*, September, 1929.

(4) **Alvis'** case was that of a woman, aged 22 years, with pneumonia, who was given 58 grains of ethylhydrocuprein over a period of three days with the result that she had no perception of light.

The pupils became widely dilated and the fundi showed thread-like arteries and veins, with retinal oedema and, in the right eye, a cherry red spot at the macula. Ten days later, she regained perception of light and 13 weeks later could count fingers and had a fairly wide field. Six weeks after this, vision of each eye was 29/75, with erythropsia and some restriction of the fields. The discs were pale and the vessels constricted or apparently empty. Histological observations in cases of quinine poisoning show degenerative changes in the ganglion cells, rods, cones and granular layers consequent on interference with their blood supply by arterial constriction and it is probable that the same occurs in ethylhydrocuprein poisoning. Uhthoff in a summary of the literature up to 1916 concluded that visual disturbances occurred in 3 to 4 per cent. of patients treated with this drug but found none who remained permanently blind, though many had contracted fields and diminished light sense.

F. A. W-N.

(5) **Green, A. S.—Ophthalmic delusions.** *California and Western Medicine*, p. 198, September, 1929.

(5) Green mentions the gross delusions held among some of the laity in regard to the eyes, but classes several views held among certain members of the medical profession as delusions also. There is the advice given that a child "will grow out of the squint," whereas 90 per cent. of all cases of strabismus need surgical intervention—before the age of 4 years if possible. Again, the fear of using prisms is a very common delusion of refractionists. That trachoma is incurable is a delusion held by many to the detriment of the treatment of cases they see. The view is expressed that practically all early cases of trachoma are curable and that over 90 per cent. of the more chronic cases can be cured. Carbon dioxide snow is advocated in the earlier stages, extirpation of the tarsi in the later stages.

That glaucoma is hopeless is a further and most pernicious delusion. Where operation is undertaken the Elliot trephining is found the best in chronic glaucoma.

That a patient with cataract must wait for operation until it is ripe is another delusion leading to much unhappiness. Where vision is above 50 per cent. in the better eye, dionine or iodide drops may be used for what they are worth. Where vision is further reduced operation is called for. Intracapsular extraction is preferred, especially in complicated cataract or in diabetes. Simple extraction even if followed by a peripheral iridectomy is condemned, as also is a section less than full half corneal. Both these proceedings lead to added risks and the use of capsule forceps rather than cystotome will further lower risks by aiding increased absorption of residual soft lens matter.

R. C. DAVENPORT.

(6) **Eales, W. H. F.— Preventible blindness.** *The Birmingham Medical Review*, June, 1929.

(6) In the Richard Middlemore Post-Graduate Lecture, 1928, **Eales** surveys some of the conditions giving rise to blindness and coming within the description "preventible" as he defined it for the purposes of the lecture. In turn ophthalmia neonatorum, phlyctenular conjunctivitis, interstitial keratitis, perforating injuries, sympathetic ophthalmitis and serpiginous ulcer of the cornea were considered in regard to their incidence, treatment and effects on visual function. For the full facts and figures the original paper must be consulted.

R. C. DAVENPORT.

BOOK NOTICES

Lehrbuch und Atlas der Spaltlampenmikroskopie des lebenden Auges. By ALFRED VOGT. Berlin: Julius Springer. 1930. Price, R.M. 178.

Volume I. Technique and Methods. The Cornea and Anterior Chamber

The slit-lamp was invented in 1912 by the late Professor Allvar Gullstrand and was used by him for the examination of the anterior portion of the eye. The first observations were made with the aid of an ordinary Zeiss field-glass, and with this inadequate equipment Gullstrand discovered that the lens was far from homogeneous. The late Professor Henker combined the slit-lamp with the Czapski corneal microscope, a great advance, which at once made the instrument of real value to the clinician. Koeppe attacked the clinical side of the question, and brought out a book dealing with the minute anatomy and pathology of the cornea. He also applied the contact-glass to the cornea and added a reflector, which, used with a special monocular microscope, enabled him to examine the whole of the vitreous and the minute details of the retina. So far the slit-lamp had been used only as a source of illumination better than that hitherto employed. The examination was the microscopy of the living eye, nothing more. Vogt now attacked the problem, and began a research which has given us a method of examination which has revolutionised our previous methods of oblique illumination. His painstaking observations have presented ophthalmology with facts regarding the anatomy and pathology of the eye, which were almost undreamt of before the advent of the slit-lamp. His work marks an epoch; he has created a new science, and those who have the privilege of reading the volume under review will inevitably come to the conclusion that there is little more to be done except to add details.

Henker modified the method of illumination, and Vogt then added the adjustable slit to the lamp. This alteration was of fundamental importance. We could now view the anterior half of the eye in *optical section*, and exact localisation was achieved. Mere surface microscopy was replaced by examination in three dimensions.

In 1921 Vogt published his first Atlas, which was at once translated into English, French, and other languages. The first edition, including the translations, was rapidly sold, and could not be obtained. Ophthalmologists all over the world have been waiting with eager anticipation for a new edition, and the work under review will not disappoint them.

The first volume which has just appeared has 300 pages and over 600 illustrations, nearly all coloured. These are on plates interleaved in the text. Our only criticism is that there are no legends. It would have been more convenient had each plate been faced with a page explaining the drawings. As it is, one has to search the text to find the description of a drawing.

The work is excellently printed on good paper, and the illustrations are real works of art. They are painted in the main by Bregenzer, the official artist at the Eye Clinic at Zürich. The price of the book, roughly £9, seems high, but it is difficult to see how such a gallery of colour could be produced for less.

The second volume which is promised shortly will be devoted entirely to the lens, and the third volume to the remainder of the eye. The complete work will cost about £27, and when it is realised that the book may be said to be the final word upon the subject, and that it is extremely improbable that anything like it will appear again, it is a good investment. The subject is so important that no competent ophthalmologist can afford to be without it as a work of reference.

The German in which the book is written is easy and direct, and can be understood by anyone with a reasonable knowledge of the language. This is by no means true of all German medical works.

In the preface the author regrets that, although most ophthalmologists own slit-lamps, the majority have not mastered the full technique, and are not able to take full advantage of the instrument. Many use the apparatus for mere surface microscopy and have not realised the possibilities of the narrow beam, of the optical section, and of exact localisation. He hopes that this text book will remedy this failure, and that the slit-lamp will become as essential to them as the ophthalmoscope.

The chief trouble the beginner experiences is simultaneously to maintain the focus of the microscope and the beam of light upon the object under observation. He must practise till he can do this automatically and without thought. We think that Vogt exaggerates this difficulty; we have found that even unqualified medical students learn the technique rapidly. They find it much easier than the use of the old-fashioned reflecting ophthalmoscope. Older men take far longer, but with a little application all difficulties disappear. It is essential to begin with a low magnification. Vogt points out that long ago attempts were made to side-track this difficulty by constructing the instrument so that the beam and the microscope were fixed to a combined focus. He says that such an instrument is "unthinkable," *ganz einfach undenkbar*; it makes it impossible to utilise the various methods of illumination, and violates the fundamental principles of slit-lamp microscopy. Those who have mastered the correct technique realise that such instruments are useless.

The first part of the volume is given to a full description of the Zeiss slit-lamp, including the arc-lamp attachment. Vogt recommends the mechanical stage mounting rather than the glass-top

table. We are of opinion that the heavier mounting is the best for hospital use, where the glass top is liable to be broken, and the microscope knocked over, but we prefer the glass top for consulting room use, and incidentally it is cheaper. The methods of adjustment are fully described, and we are warned to keep the slit free from fluff and hairs which destroy the perfection of the beam and make accurate work impossible. The slit is delicate and an ordinary sable paint brush should be used for the purpose. It is not necessary that the room should be quite dark for ordinary examinations. The finer points are seen only when the observer's eye is dark adapted. Both patient and surgeon should sit on stools adjustable for height. Exact surface measurements can be made with the micrometer eyepiece, and if a goniometer is used to measure the angle between the axes of the lamp and microscope, anteroposterior measurement can be made by the usual trigonometrical methods. The micrometer drum on the microscope enables us to estimate the depth of the anterior chamber, the thickness of the lens, and so forth. Then follows the important chapter on the varied methods of illumination. The chief are direct focal illumination, examination by transmitted light, and the use of the area of specular light. Employment of mirror light allowed Vogt to observe the endothelial cells of the cornea, and the epithelial cells of the anterior capsule. Exact localisation by the use of the narrow beam is carefully described and merits the closest study. Measurements with the micrometer eyepiece are very valuable to the clinician in so far as they relate to surface estimation. It is useful to have a record of the extent of a corneal infiltration, or of a lens opacity, but when triangles have to be solved the work becomes academical and unsuited to the everyday work of the clinician. Here the method of estimation with the narrow beam is of vital importance, and is in constant use. With the narrow beam the expert can by simple inspection localise the exact position of say a foreign body in the cornea, or of an opacity in the lens. Such estimations are often necessary and are made in a few minutes, with an exactitude quite sufficient for clinical use. Study of the endothelial cells may seem to have little practical value, but when we realise that dimming of the outlines of these cells may be one of the first signs of inflammation, then such observations are seen to have a real practical significance.

In the original atlas Vogt described a bedewing, oedema, of both the anterior and posterior surfaces of the cornea. He has now come to the conclusion, and we feel that he is correct, that there is no such thing as posterior oedema. The appearance suggesting a posterior bedewing is due to deposit of lymphocytes on the endothelium. Such are seen in the normal eye, forming Lüssi's line, which is common in children. In early inflammation the whole corneal

surface may be covered, and the impression of an oedema is conveyed to the observer.

The *Special Section* of the book begins with the normal cornea and the changes that occur with advancing age. A dark adapted eye, with careful focusing, can differentiate the *epithelium* from Bowman's membrane, and naturally any separation of the epithelium is at once obvious. The *endothelium* with the Hassal-Henle warts is shown in Illustration 47, and in 52 the normal appearance of the *corneal nerves* is demonstrated. These lose their sheaths as they enter the cornea, but occasionally they are preserved for some distance into the clear cornea, a condition comparable with opaque nerve fibres in the retina. The nerves are to a large extent confined to the anterior two thirds of the cornea. They generally divide dichotomously, but modifications are seen, nodules on the nerve fibres and webs at the points of division. The *visible circulation* of the blood in the limbal vessels is seen only when the column is interrupted, and when transmitted light is used. Plate 12 is devoted to *arcus senilis* and its variations. Even in youth an *arcus juvenilis* is not infrequently seen with the narrow beam.

A *peripheral senile furrow* is seen in Figure 101, occupying about one third of the cornea. This is apparently faced by a corresponding furrow on the posterior surface of the cornea, but this is merely an illusion caused by the irregular refraction of the surface furrow.

The superficial *lines* which develop in the senile cornea are fully described and illustrated. The so-called *Stähli's line* was first described by *Hudson* using ordinary oblique illumination and the common loupe. It takes a variety of forms as seen on Plates 15 and 16. A *deposit of pigment* on the posterior face of the cornea is very common in advancing age, many varieties have been described as occurring deep in the corneal parenchyma. Careful use of the narrow beam localises them on the posterior surface of the cornea. Vogt regards *Krukenberg's spindle* as a special modification of the ordinary deposit on the endothelium. It may take a variety of forms, and has been described in the horizontal position and inclined. *The beaten-silver cornea, cornea guttata* is held to be associated with Fuchs' epithelial dystrophia. Vogt has had the opportunity of making a microscopic examination of an eye with this affection, and microphotographs are given. The central axial portions of the cornea show prominences of Descemet's membrane reminiscent of the Müller-Henle warts, but they are found axially, never in the periphery, whereas the warts are not so localised.

Cornea farinata, in which the posterior surface seems to be covered with flour-like dust is as far as we know a new discovery. Its nature is made abundantly clear by a fine drawing. Another new condition is the *white limbus girdle*. This degenerative process is not easy to see and is visible only by retro-illumination.

Groenouw's disease, *nodular keratitis*, is illustrated in its various forms on Plates 29 and 30. This is a hereditary degeneration situated in the superficial and medial layers of the parenchyma.

Parenchymatous crystalline degeneration of the cornea is found in middle age and in the senile period. It is not associated with any inflammation or vascularisation, is slowly progressive, and characterised by the formation of crystalline needles which are most numerous in the advancing peripheral border.

The appearances seen in *keratoconus* are now well known. Fleischer's ring and the well-marked striae are well shown in numerous drawings. The endothelial cells are especially prominent

and are seen over a wide area. Corneal nerves are large and greatly increased in number.

In ***pseudo-sclerosis*** (Kinnier Wilson's disease) the liver shows a characteristic sclerosis and in the region of the corpus striatum there is neuroglial proliferation and degeneration.

The first case was described by Westphal in 1872 and called by him pseudo-sclerosis. In 1912 Kinnier Wilson described a similar, perhaps identical, syndrome, to which he gave the name of progressive lenticular degeneration. In 1902 Kayser, and in 1903 Fleischer, both of Tübingen, described a pigmented corneal ring in cases of pseudosclerosis. The latter also found on autopsy peculiar pigmentation of various organs. Rumpel and Söldner found that the pigment in the kidneys was due to silver. Söldner discovered traces of antimony and a large amount of copper in the liver. This discovery was not confirmed by later investigators, but when the celebrated case, Elise Utzinger, who had been kept in the medical wards under Nägelli and others for over forty years, died, Vogt had a careful chemical examination made by the most modern methods. In this case and in another similar patient the liver contained an enormous amount of copper, and the kidney considerable silver.

In some cases of pseudosclerosis a genuine copper cataract of the typical sunflower type has been present in both eyes, although no copper had entered the eye or had been used in the treatment. Vogt has seen cases in which pigmentary changes in the region of Descemet's membrane have been present in eyes that have been treated with colloidal silver preparations and he has succeeded in inducing similar pigmentation by instillations of argyrol. His drawings are similar to a case published by the reviewer in this Journal, Vol. XIII, page 401. There can be no doubt that this hitherto unexplained case was due to argyrol which had evidently been employed at some time or other in the treatment. The course of this case was exactly that described in Vogt's experimental cases and the bright cyanine pigmentation was the same. Finally Vogt has treated sections of the cornea showing the Fleischer ring with potassium cyanide and has dissolved out the coloration. He thinks, and those who read his remarks on the subject will agree with him, that the Kayser-Fleischer ring is caused by diffraction phenomena due to the presence of very fine particles of metallic silver in the cornea. Investigations at Vienna by Gerda Laski, show that such particles can and do produce colour effects. The sunflower cataract is certainly due to the presence of copper in the blood, for this form of cataract is absolutely typical and is caused by nothing but copper in the eye. Vogt suggests that Kinnier Wilson's disease is due to a failure of the liver to eliminate metal absorbed in food, or even that it collects and stores silver and copper. This causes a toxaemia which is responsible for pseudosclerosis.

Keratitis epithelialis. It is impossible to classify the many conditions seen with the slit-lamp which fall under this heading. Several varieties are pictured in Plates 41 and 42. It is not even known whether they are degenerative processes or inflammatory; they can be described and named only from their anatomical appearance.

Interstitial keratitis. The drawings of the many varieties of keratitis profunda are worthy of careful study. The narrow beam shows the enormous thickening that is present in the acute stages and later the thinning and ectasias that may follow. Typical of the congenital syphilitic type is the sclerosis of the posterior layers of the cornea. This may reflect light even more vividly than the corneal surface, and taken with the presence of deep vascularisation leaves little doubt as to the nature of the original disease.

The anterior chamber. The slit-lamp is of great value in determining the depth of the chamber. Often by ordinary examination there would appear to be no chamber left, say after an operation for glaucoma, but the slit-lamp shows that at the periphery and in the

pupil area there is still a collection of aqueous. Examination of the aqueous with the slit-lamp may detect the first signs of inflammation. The normal fluid is almost optically inactive, but when it becomes loaded with albumin the beam becomes visible as it traverses the anterior chamber, just as the headlights of a car are obvious to the driver in a fog. The density of a beam may be taken as a measure of the albumin content of the aqueous. Again, particles in the aqueous show a streaming motion in the convection current in the aqueous, rising at the back and falling in front. As soon as the albumin content reaches a certain concentration the particles come to rest. The re-establishment of the convection current is the first sign that the inflammation is abating. After operations and accidents the vitreous may enter the anterior chamber. The extent and nature of the prolapse can be studied with the slit-lamp.

The scope of the work is so enormous that we have been able to touch upon but a few of the subjects, but we hope that we have said enough to convince our readers that the book is one that no ophthalmologist can afford to be without. Even for those who cannot read German, the illustrations will be of the greatest value.

T. HARRISON BUTLER.

Ofthalmologicky Sbornik. Svazek V, 1930. Collection of Papers read before the Fifth Congress of the Czechoslovak Ophthalmological Society, 1930. Published by the Society, Prague.

This volume of ***Transactions***, containing 41 communications, is, as usual, supplemented by summaries of the papers in English, French and German. The summaries are very brief and do not enable the reader to gain more than a scanty idea of the contents or value of the papers. The number and variety of the communications bear witness to the zeal and activity of the members of the Society.

Glaucoma in some of its aspects is the text of 19 of the 41 papers. *Z*-Zichova induced acute inflammatory glaucoma in a rabbit by injecting trypaflavin into the anterior chamber. Examination of the eye showed oedema of the cornea, iris and ciliary body, contracted arterioles in the iris due to swelling of the endothelium, with dilated and blocked veins.

Slavik reported 13 cases of hydrophthalmos in which varied operative measures were almost invariably unsuccessful. Franta examined the blood serum for alteration in the quantity of salts of calcium and potassium, in 16 cases of primary glaucoma and 16 controls. No abnormality was found in the calcium content, but in all cases a slight diminution of potassium content was detected. Slavik's results from cyclo-dialysis are given: 127 operations on 97

patients. In 69 cases of simple glaucoma good results were obtained in 60·9 per cent; in 22 cases of chronic inflammatory glaucoma the result was good in 31·8 per cent. The remainder were examples of secondary glaucoma. Gala, in 438 cases of primary glaucoma found myopic refraction in 1·6 per cent; all but one occurred in males. One man, aged 44 years, had myopia of 7 – 8D. Knobloch gives his findings as to the aetiology of scleritis, in 65 cases seen during a period of 10 years. In 79·3 per cent. tuberculosis was deemed to be the cause; in 11·1 per cent. the cause could not be determined; other probable causes such as syphilis, staphylococcus infection, uricacidaemia, are credited with the remaining cases. In view of the great preponderance of tuberculosis cases, the author counsels treatment on anti-tubercle lines in all cases of obscure causation. Klauber reports the successful treatment of a "typical rodent ulcer" of the cornea by 5 per cent. gold chloride and tannin. The remaining summaries are of papers on Progressive Ophthalmoplegia Externa, Therapy of Pituitary Tumours, Long Retention of Intra-ocular Foreign Bodies, Orbital Abscess, Ethmoidal Mucocele, etc.

CORRESPONDENCE

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

GENTLEMEN,—The following notes of a case of high astigmatism, which came for refraction to Moorfields Hospital a few weeks ago, may be of interest. The case was under the care of Mr. Neame to whom I am indebted for permission to publish it.

Mr. C. S., aged 30, commercial traveller, complained of "distant vision not so good as formerly." According to the old notes of January, 1928, he had vision

R. $\bar{c}$ – 1·0 D.sph. $\bar{c}$ +1·0 D.cyl. 160° → 6/9
L. $\bar{c}$ – 1·5 D.sph. $\bar{c}$ +1.0 D.cyl. 20° ← 6/36

I found vision

R. $\bar{c}$ – 2·0 D.cyl. 90° ↓ 6/6 most
L. $\bar{c}$ + 5.0 D.sph. $\bar{c}$ – 10·0 D.cyl. 97½° ↓ 6/9

It was possible, by naked eye observation of the corneal image of a window, to tell which was the meridian of greater curvature.

Yours truly,

F. C. WILSON.

NOTES

Royal London Ophthalmic Hospital, Annual Dinner.

THE annual dinner of past and present students of the Royal London Ophthalmic Hospital will be held at the Langham Hotel, on Thursday, February 12, at 7 p.m. for 7.30 p.m. The Chairman will be Mr. A. A. H. Sinclair, of Edinburgh. Those intending to be present are asked to communicate, as early as possible, with Mr. Rupert Scott, 70, Harley Street, W.1. The price of the ticket is 15/- (excluding wine).

* * * *

British Medical Association.

THE 99th annual meeting of the British Medical Association will be held at Eastbourne from July 22 to July 24. The President of the section of ophthalmology is Mr. E. E. Maddox.

FUTURE ARRANGEMENTS

1931

February 6.—North of England Ophthalmological Society. Joint meeting with the Scottish Ophthalmological Club at Newcastle-on-Tyne.

February 6.—Midland Ophthalmological Society, at Leicester.

February 13.—Section of Ophthalmology, Royal Society of Medicine.

March 6.—North of England Ophthalmological Society, at Liverpool.

March 13.—Section of Ophthalmology, Royal Society of Medicine. (Clinical Meeting).

March 20.—North of England Ophthalmological Society, at Sheffield.

March 31.—Midland Ophthalmological Society, at Birmingham.

April 23-25.—Annual Congress of the Ophthalmological Society of the United Kingdom, in London.

June 5.—Midland Ophthalmological Society, at Cheltenham.

June 12.—Section of Ophthalmology, Royal Society of Medicine. (Annual Meeting).

October 6.—Midland Ophthalmological Society, at Birmingham.

December 1.—Midland Ophthalmological Society, at Birmingham.

CONTEMPORARY OPHTHALMIC LITERATURE

American Journal of Ophthalmology. *December, 1930.*

POE. Kayser-Fleischer ring in the cornea in a case of Wilson's disease.
FRIEDMAN and RÖTTH. Argyrosis corneae.
SWAB. Latent heterophoria as noted after prolonged monocular occlusion.
DELANEY. The light sense as tested by the photometric glasses of Tscherning.
FRY. Congenital total colour blindness.
BLACK. The Guist speculum.
DERBY. Personal experience in the management of cataract.
SALIT. Calcium determinations on cataractous human lenses.
SMITH, BARKAN and BARKAN. Vasomotor glaucoma.
SPRATT. Intra-ocular foreign bodies.
JAMES. Inclusion blennorrhea.
REED. Oculist, optician, and patient.

Archives of Ophthalmology. *November, 1930.*

GONIN. The treatment of detached retina by searing the retinal tears.
BROWN and KRONFELD. The acuity of binocular depth perception in hemianopia.
RILEY. The central nervous system control of the ocular movements and the disturbances of the mechanism (to be concluded).
FINLAY. A modification of Gonin's surgical method of treatment of detachment of the retina.
——— Atypical (upward) coloboma of the choroid: clinical description.
DAVIS and SMITH. The rôle of lens antigen and uveal pigment in the production of hereditary anomalies of the eye.
AEBLI. Optic atrophy in Paget's disease.
BEDELL. The significance of some changes in the fundus vessels.
KNAPP. Small tumour of the iris which caused secondary glaucoma: relation of melanoma to sarcoma of the iris.

Annales d'Oculistique. *November, 1930.*

EMILE VALUDE (1857-1930).
GALLEMAERTS. Cyst of the cornea.
DUPUY-DUTEMPS. Blepharoplasty by a tubular graft from the neck.
CHIAZZARO. Notes on the diplobacilli of Morax and Petit.
KALT. Exudative retinitis without vascular change.
THEODOR AXENFELD (1867-1930).
ALLVAR GULLSTRAND (1862-1930).

December, 1930.

J. B. COPPEZ (1840-1930).
DUFOUR. The astigmatism of a pencil of light refracted by a spherical lens of one dioptre.
——— The aberration of a one dioptre sphere. The relation between the aberration and the astigmatism.
MARX. Simultaneous recording of the intra-ocular tension and the tension of the cerebro-spinal fluid.
PUSCARIU. Plasmoma of the conjunctiva.
DE ANDRADE. Some researches on trachoma. Biomicroscopical and histo-pathological study.

Archives d'Ophtalmologie. *December, 1930.*

DE LAPERSONNE. Jean Coppez.
PASCHEFF. Chronic hyperplasia of the conjunctiva and true trachoma.
CHARAMIS. On a case of unsuspected juxta-papillary sarcoma of the choroid.
COLRAT. Episcleral and conjunctival vasodilatation following mumps.
International Association for the prevention of blindness.

Archiv für Ophthalmologie. *November, 1930.*

PILLAT. Bacteriological findings in praexerosis corneae.
HERZAU. On the clinical aspect of unilateral and bilateral ophthalmoplegia of peripheral origin.
POLLAK. Investigations on a surviving human eye.
JECKLEN. Bilateral total ophthalmoplegia following metastatic sarcoma of the sphenoid.
BLATT. Crises of accommodation in tabes.
OHM. On nystagmus.
IMAI. Experimental investigation of bone formation in the eye.
CABALLERO. Experimental clinical investigation on the question of glaucoma.
POOS. On the part played by the two antagonists in the movements of the pupil in different reactions.

Klinische Monatsblätter für Augenheilkunde. *November, 1930.*

HIRSCH. On tumours of the hypophysis and their treatment.
PETERS. Clinical notes on the question of recurrent erosion and herpes of the cornea.
JAHNKE. On the investigation and treatment of the apparently normal conjunctiva before intra-ocular operations.
DEUCHLER. Trichophyton cerebriforme in the conjunctival secretion.
MEERHOFF. Treatment of xanthelasma with thorium X Degea.

December, 1930.

SIEMUND. A case of lymphomatous disease of the conjunctiva.
MARX and QUARTERO. Lowering of the intra-ocular pressure after contusion of the eye.
CHARLIN. Aetiology of papilloedema.
V. PELLATHY and SCHNEIDER. The treatment of conjunctival and corneal inflammations, particularly trachoma, with sodium hydrocarbonicum on the ground of the investigation of the pH value of the conjunctival secretion.
GUTMANN. Light treatment of eye diseases with the focal ultra-violet lamp, model 2.
PFLIMLIN. On the treatment of alcohol-tobacco amblyopia with nitroskleran.
PROCKSCH. On Imre's treatment of disease of the fundus with amylnitrite.
SZÉKELY. On the discussion of rare fundus conditions.
HOLTH. The revived use of Galen's corneal stain of ferric sulphate and tannin must be given up.
EISNER. Note on anomaly of growth of the cilia.
LÖWENSTEIN. Iodipin treatment for discharges from the lacrimal sac.
WIECHMANN. Clinical investigation of the connection between osmosis, blood pressure and intra-ocular tension.
BUSACCA. Detachment of the zonular lamella, the lens capsule or a deep membrane. (An addition to the work of Rehsteiner "Research into the knowledge of lens membrane glaucoma.")
ASCHER. Ring-shaped coloured contact glasses in albinism.

Zeitschrift für Augenheilkunde. *December, 1930.*

KNEPPER. On accidental injuries in childhood.
RAUH. On the treatment of glaucoma with glaucosan.
HOLLÓS. Resection of the orbicularis in paralytic ectropion.

MALKIN. On primary degeneration of the cornea.
RACHMANN. Injuries with splinters of iron, from the municipal eye clinic in Essen, during the years 1913-1930.
HAASS. Note on the treatment of irritative formations especially with protasin and perprotasin.
MEISSNER. On the instruction of the weak sighted in blind asylums.

Archivio di Ottalmologia. *September, 1930.*

Meeting of the Council of the International Association for the Prevention of Blindness.
FEDERICI. On the surgical cure of convergent strabismus of high degree.
FERRARI. Monocular retrobulbar neuritis following sphenoidal sinusitis cured by the removal of the middle turbinal following the trephining of the sphenoidal sinus. Clinical contribution.
MAZZI. Ocular symptoms of meningitis.

Anales de la Sociedad Mexicana de Oftalmologia y Oto-Rino-Laringologia.

May-June, 1930.

VELÉZ. Complementary notes on the onchocercosis of Mexico.
MCREYNOLDS. The crystalline lens system.
AVALOS. A case of diabetic cataract recognised as of specific origin.
VILLASEÑOR. Foreign bodies in the eyes of railway workers.
GRANDE. Tuberculin therapy in ophthalmology.

July-August-September, 1930.

Campaign against Leprosy.

History. LANDA. Leprosy. Historical interest.
Symptomatology. VÉLEZ. Preamble to work of Dr. Santiago Ramírez.
——— RAMIREZ. Description of the precautions for the avoidance of leprosy.
Department of Public Health. Precautions for the avoidance of leprosy.
Prophylaxis. VELÉZ. Precautions in cities for the avoidance of leprosy.
Diminution of Leprosy. LEE. Why did leprosy diminish in Norway?
The World of the Leper. HEISSER. The obligation of the world toward the lepers.
SALDANA. The bacteriology of leprosy.
Sanitation of Leprosy. Federal regulations of the prophylaxis of leprosy.
Department of Public Health. Circulars and pamphlets.

Revista Cubana de Oftalmologia y Oto-Rino-Laryngologia.

December, 1930.

FFRNÁNDEZ. Impermeability of the lacrimal passages.
RIVOIRE. Cataract and calcium metabolism.
AVALOS. A case of diabetic cataract recognised as of specific origin.
CASTRESANA. Gold salts in ocular therapeutics.

Revista Oto-Neuro-Oftalmologica y de Cirugia Neurologica.

January, 1931.

LACARRERE. Self-registering photocampimeter.